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THE PRESENT STORY ON BATTLE CASUALTIES FROM KOREA

A Six Months' Study

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MUCH interest has been expressed concerning the results of medical policy and management of battle casualties and other causes of disability in the Far East since the onset of the Korean action. The results have been excellent, and consequently it is deemed appropriate and timely that a statement of performance to date be made. It is difficult during any such conflict to obtain all available data since much of it is classified, and it is never possible until a considerable time after the conclusion of any conflict to summarize and weigh the entire mass of material and information which has resulted. Nonetheless, it has been possible for the purposes of this report to present information on evacuees from the Far East covering a 6 months' period, taken for the most part from September 2, 1950 through February 28, 1951, which will illustrate the work that is being and has been done. In some instances it has been possible to include material from a somewhat longer period of observation, when that extended period could bring the information to a more current date without disturbing the comparisons and material of the special study period.

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The initial development of medical service for Korea in June 1950 presented the difficulties inherent in a sudden onset of hostilities for which anticipatory preparedness could not be made. Of first priority was the need for additional skilled personnel overseas to care for battle casualties and fortunately we had immediately available the fine group of young regular Army officers who were enrolled in the military and civilian residency training program which had been in existence since 1947. These young physicians with the splendid help of the medical officers already in the Far East and the veteran reserve officers who volunteered for service are largely responsible for the outstanding results being obtained. A limited field service was able early to support the combat forces in a superior manner since the conflict is in an area immediately adjacent to a well organized base, namely Japan, where an unmolested Army hospital system housed in permanent facilities has been in operation for several years. The close proximity of the Korean incident to World War II also resulted in early and effective application of the many lessons learned in the latter conflict. We were fortunate in having on duty an appreciable number of officers well experienced in war surgery, who were not only available to perform

professional services themselves, but also able to instruct and supervise those younger men without prior war service, mature judgment, or firsthand experience in the application of the lessons from the previous war. In spite of obvious deficiencies and the usual early errors in surgical judgment and experience, the overall understanding of war surgery and the early application of accepted methods of management have resulted in a performance which to date surpasses or equals even the exceptionally fine record compiled during World War II. It should be a matter of pride to all of those who served in the past conflict that the policies, procedures, and principles which they so steadfastly advanced and taught have borne such good and early fruit in the Korean campaign.

Not only was there a shortage of medical personnel in the theater and a lack of wide war-surgery experience among the majority of them, but also the Korean terrain posed a problem in the maintenance of supply and of early evacuation. The conflict has been one of maneuver with a constantly shifting battle line. A certain lack of clear-cut military objectives and overall purpose made long range medical planning difficult, although the latter became, of course, intimately involved with our long range preparedness program within the United States. Nevertheless, it is possible to state without reservation that the sick and wounded of the Far East are continuing to receive excellent care, and that the medical talent on duty in the theater is being utilized to the fullest.

Beginning with the evacuation of the first wounded from Korea through Japan, we have made continuing surveys as to the physical condition in which patients have been received and have been continuously alert to improve forward medical care, wound management, methods and timing of evacuation, and all other problems involved in good medical service. The care of the wounded in any battle zone must always be influenced by the conditions and circumstances that govern the prevailing overall tactical situation, and this is reflected along the entire line of evacuation affecting eventually the type of care required and rendered in the general and specialized treatment hospitals in the United States. Details of local topography in the country in which the conflict is waged bear directly upon this matter, and in Korea we have been faced with problems of the greatest magnitude and difficulty. It is erroneous to assume, however, that the surgery of war is entirely molded by these concessions, and that military surgery must perforce be regarded as a crude departure from surgical standards. We have recently reviewed and rewritten our *Technical Bulletins, Medical, on Management of Battle Casualties, Resuscitation And Early Care of the Wounded* for the expressed purpose of providing guidance and instruction to our personnel in the field. The steady pressure exerted upon all facilities by the constant inflow of wounded personnel is admittedly contrary to ideal surgical management of the individual since it may require too early evacuation. Differential adjustment must be made on the basis of the type and severity of the injury, the complications, probable duration of disability, correct triage, and considerations of saving of life, ultimate disability, deformity, and delayed recovery. This problem is complex and is to be borne in mind by all who are interested in the care and evacuation of mass casualties, whether military or civilian.

Since the fall of 1950, the hospital bed capacity in the Far East has been increased and the ability to hold casualties thereby extended. In this way, together with increased staffing, many that formerly would have been evacuated now remain and are screened, treated, evacuated, or returned to duty within the theater. In Table I data are presented for the period July 1, 1950 to March 2, 1951 showing the disposition of patients.

The high percentage of casualties evacuated to the United States resulted from our authorized 120 day maximum overseas hospitalization policy, and the early evacuation of many cases for specialized treatment.

During the last quarter of 1950 especially, the case load in the Far East was far in excess of the available bed capacity there, and consequently it was necessary to utilize rapid air evacuation to the United States in an increased amount in order to maintain adequate space for new and serious injuries. It was dur-

ing this period that many patients with minor wounds were evacuated in order to make room in Japan for the seriously wounded nontransportable patients. This period quickly passed and we now have in Japan, in addition to the active treatment hospitals, two convalescent centers to take care of the need for sufficient overseas beds to retain the lightly wounded and return them to duty without draining away manpower by nonselective evacuation. Doubtless some wounded who could be returned to duty in the theater have been, and are being, evacuated under the rotation of combat troops policy now in effect. Top priority in evacuation to the United States is now given all major battle wounded when logistically possible and not detrimental to patient.

Furthermore, a reduction in the time from wounding to surgery has resulted in the saving of life, the saving of extremities, the minimizing of deformity, and the shortening of convalescence, as well as the avoidance of undue dissipation of a fighting force by excessive loss of manpower through delayed transportation. That this has been given careful thought and definite action is apparent from the fact that the Japan Logistical Command has been able to return to duty the equivalent of one full division at combat strength every 2 months. This is indeed a splendid achievement and serves to indicate the high standard of treatment and facilities available in the Far East.

Major General Raymond W. Bliss, while serving as the Surgeon General of the Army, made personal inspection tours of all medical installations in Japan and Korea in September 1950 and April 1951. His report contains the following statement which serves as an excellent summary of our policy and planning in that theater: "The problems have received and will continue to receive the most serious consideration and planning by all concerned personnel, both professional and administrative. I feel that all factors in medical care are receiving proper consideration, management and attention; consistent with combat policies and the highest medical standards. I am convinced on the basis of personal observation and growing statistics that the results in and from Korea constitute a bright chapter in the history of Military Medicine."

TABLE I.—SELECTED DISPOSITIONS OF PATIENTS AS A PERCENTAGE OF ALL ADMISSIONS TO MEDICAL TREATMENT FACILITIES DURING THE SAME PERIOD—JAPAN AND KOREA, COMBINED (JULY 1, 1950 TO MARCH 2, 1951)

Specified category is percentage of total admissions[1]

Disposition	Disease cases per cent	Nonbattle injuries per cent	Battle casualties per cent
Returned to duty in Japan or Korea[2]	90.8[2]	73.0[2]	65.3
Died in Japan or Korea	0.1	1.0	2.6
Evacuated to United States	3.8	11.1	28.6

[1]A small proportion of the dispositions included represent disposition of cases on hand at the beginning of the period; these, however, are not separately identifiable.

[2]Also included in this category are certain cases treated on a duty status but included as admissions because they were "carded for record only." Since such cases were treated on a duty status they are included with those returned to duty.

It is possible at this time to release the following information for the period of 6 months. This material has been compiled in various ways and serves well to illustrate the prodigious effort made by the medical services to provide early and adequate treatment to the battle wounded and all other casualties. In order to conserve space, this material is presented in relatively brief summary form, with tables of data, and presented under various pertinent sections. It is not possible at this early date for us to submit the entire story of the conflict, nor to consider every medicomilitary phase, but we do believe that the present story as given herein is appropriate and timely.

GENERAL CONSIDERATIONS

During the period September 2, 1950 through February 28, 1951 a total of 7,039

TABLE II.—BATTLE CASUALTIES SUSTAINED BY ARMY PERSONNEL, NOTIFICATIONS OF WHICH HAD BEEN MADE TO EMERGENCY ADDRESSEES THROUGH FEBRUARY 23, 1951

	No. cases	No. cases
Total battle casualties		41,951
Total battle deaths		7,017
Killed in action	6,274	
Died of wounds or injuries received in action	658	
Died while prisoner or in missing in action status	85	
Wounded or injured in action, excluding those shown as battle deaths		25,940
Missing in action or captured, excluding those shown as battle deaths		8,994

TABLE III.—EVACUEES RECEIVED AT HOSPITALS OF DEFINITIVE TREATMENT IN THE UNITED STATES BY TYPE OF SPECIALIZED TREATMENT, BY TYPE OF PERSONNEL, WITH AN ADDITIONAL BREAKDOWN BY TYPE OF CASE FOR ARMY PERSONNEL ONLY

Type of specialized treatment required[1]	Type of personnel					
	Army				Air force	Civilian
	Type of case					
	Total	Battle casualty[2]	Non-battle injury	Disease		
Totals	12,908	7,041	1,912	3,955	369	75
Amputation	169	147	22	0	2	0
Arthritis	160	1	3	156	36	0
Blind	6	3	0	3	0	0
Deaf	71	8	1	63	7	0
General medicine	1,458	151	127	1,180	70	27
General surgery including orthopedic surgery	7,448	5,552	1,180	716	59	21
Neurological	138	18	4	116	19	1
Neuropsychiatric	1,033	25	5	1,003	91	13
Closed ward	(353)	(12)	(2)	(339)	(54)	(2)
Open ward	(680)	(13)	(3)	(664)	(37)	(11)
Neurosurgery	272	179	28	65	12	0
Neurosyphilis	2	0	0	2	0	0
Ophthalmological surgery	306	164	8	134	13	0
Plastic and maxillofacial surgery	27	12	10	5	0	0
Rheumatic fever	4	0	0	4	0	0
Skin disease	59	1	1	57	19	1
Thoracic surgery	17	15	0	2	1	0
Cold injury	393	10	383	0	1	0
Tropical disease	1	0	0	1	0	0
Tuberculosis	144	2	2	140	32	1
Vascular	26	0	0	26	2	3
Not specified	1,174	754	138	282	5	8

[1]Based on first diagnosis only, i.e., condition for which evacuated.
[2]The battle casualties reported as requiring certain types of specialized treatment such as general medicine (151), neuropsychiatric (25), skin disease (1), and tuberculosis (2) presumably represent cases hospitalized for battle injury or wound and for disease concomitantly, with the disease condition being reported as the primary cause of *evacuation* although the case was classified as a battle casualty on the basis of primary cause of *admission*. The same comment is applicable to the 10 battle casualty cases reported as requiring specialized treatment for cold injuries.

Army battle casualty patients were received at hospitals for definitive treatment from Korea and Japan. In order to provide a framework of reference, certain data are presented concerning the battle casualty experience in Korea. These data are based on releases made by the Department of Defense and, from a time viewpoint, represent the cumulative numbers of casualties concerning which notifications to emergency addressees have been made as of the date shown. The date for the following data has been selected arbitrarily as February 23, slightly earlier than the date used for the evacuee data. The battle casualty evacuee figures represent cases received at various hospitals in the United States through the 28th of February, and these cases, therefore, originated out of the total group of casualties occurring through some earlier date (Table II). It is obvious from consideration of the casualty data that the 7,039 battle casualty evacuees presented in this paper represent only a fraction of all casualties sustained. And even though the incomparability of time periods prevents any precise calculation, it appears from the number of cases reported as wounded or injured in action that something less than one-third of them are evacuated to the United States. As a matter of fact, this gross estimate is consistent with other data which indicate that of all battle casualty cases admitted to medical treatment facilities, approximately two-thirds have been disposed of by return to duty in Japan or Korea (Table I).

The patients evacuated to the United States came by ship (9.1 per cent) and by air (90.9 per cent). The average number of days per case *prior to* evacuation was virtually the same whether the man was sent by ship (20.6 days) or by air (20.8 days). However, the time these patients spend in actual travel status was 11.5 days by ship, but only 3.4 by air. The latter group, contrary to the ship evacuees, required an average of 1.4 days of stopover at a hospital facility outside the zone of interior while en route.

In order to provide some basis for comparison, Table III is included which shows the total patients evacuated from *all overseas theaters*, including the 11,638 Army casualties from Korea, the type of specialized treatment indicated, the type of case origin, and personnel comparison with Air Force and civilian persons treated by the Army. Data on the

Air Force, Navy, and Marines are not available for this study.

Of the 12,908 Army evacuees from *all* overseas theaters, we examined the records to determine what numbers had undergone some type of surgical operation incident to their disability (Table IV).

The noted incidence of prior surgical procedures refers primarily to major or definitive work performed and does not necessarily include minor or first aid measures. These latter patients would not be evacuated as such, although it is known that such case types exist among the other casualties evacuated by virtue of a major illness or other disability which took priority in consideration for evacuation. We have no figures available on usage of blood, but it is of interest to note that 64,091 pints of whole blood were procured for use during the period studied.

The specific type of surgical operative procedure performed prior to evacuation to United States during the period September 2, 1950 to April 30, 1951 is presented in Table V.

The type of surgical procedure performed is subdivided to present the number of operations performed in each surgical specialty. Study of these data definitely indicate that the "surgical work load" in each specialty is impressive. The somewhat equal distribution of surgery in each of the surgical specialties should have tremendous influence in formulating the professional training program of our future military surgeons. The policy of having specialty surgery performed by the highly qualified specialist in the forward areas is ideal, however, the number of specialists required would make this ideal condition prohibitive. The obvious alternative would be to develop military surgeons, well trained, in the specialty care of all types of trauma incident to warfare.

Information furnished from these data, from periodic consultant reports, and from theater surgeons has enabled the Medical Service to assign medical officers efficiently, not only in appropriate numbers but in the specialty required (Table VI).

Table VI is of special interest in that the data also include the time period March 1, 1951 to April 30, 1951, or a 60 day period not included in all other data presented in the study period. Approximately 36 per cent of all evacuees (16,711) were subjected to an operative procedure.

TABLE IV.—ARMY EVACUEES RECEIVED AT HOSPITALS OF DEFINITIVE TREATMENT IN THE UNITED STATES BY TYPE OF CASE AND INCIDENCE OF PRIOR SURGICAL PROCEDURE (SEPTEMBER 2, 1950 TO FEBRUARY 28, 1951)[1]

Type of case	Number	Percentage of all Army evacuees	Percentage of each type of case
Total	12,908	100.0	100.0
With operation	4,129	—	32.0
Without operation	8,779	—	68.0
Disease—Total	3,955	30.6	100.0
With operation	163	—	4.1
Without operation	3,792	—	95.9
Nonbattle injury—Total	1,912	14.8	100.0
With operation	1,255	—	65.6
Without operation	657	—	34.4
Battle casualty—Total	7,041	54.6	100.0
With operation	2,711	—	38.5
Without operation	4,330	—	61.5

[1]Based on first diagnosis only which represents the primary cause of evacuation. Operations performed for the second diagnosis are omitted here.

PERSONNEL

During the period of study, there was an average of 1,000 Army medical officers in the Far East to care for the troops in Korea and Japan, the dependents of personnel, and the prisoners of war. There was 1 medical officer for every 73 hospital patients in Korea, and 1 for every 31 in Japan Logistical Command. Hospitals functioning in the communication zone in Korea never exceeded 91 per cent of their authorized strength in medical officers while combat units always functioned with less than 80 per cent. At no time during the conflict has the number of medical officers assigned been considered in excess of the combat situation (Table VII).

Every precaution has been taken to utilize each medical officer within his specialty or capability, insofar as the demands of the tactical situation permit. The overall ratio of physicians to troops throughout the service has been reduced to 3.5 per thousand, in con-

TABLE V.—EVACUEES RECEIVED AT HOSPITALS OF DEFINITIVE TREATMENT IN THE UNITED STATES BY TYPE OF OPERATIONS PERFORMED (SEPTEMBER 2, 1950 TO APRIL 30, 1951)

Type of operation performed	Total evacuees	Type of case		Total Army
		Battle casualty	Other	
Grand total—All operations	7,154[1]	5,746	1,408	7,090
Neurosurgical operations	357	281	76	351
Craniotomy or craniectomy	198			
Other neurosurgery	159			
Surgery of eye, ear, nose, or throat	331	282	49	327
Enucleation or evisceration	102			
Other eye surgery	160			
Surgery ear, nose, or throat	69			
Oral surgery	116	100	16	116
Reduction of fracture of mandible	83			
Other oral surgery	33			
Thoracic surgery	232	194	38	226
Thoracentesis	125			
Other thoracic surgery	107			
Abdominal surgery (incl. proctologic)	454	365	89	451
Colostomy	107			
Laparotomy	106			
Other abdominal surgery	241			
Genitourinary, gynecological, and obstetrical surgery	119	65	54	117
Genitourinary surgery	116			
Obstetrics and gynecological surgery	3			
Amputations or disarticulations	409	346	63	404
Lower extremity, major amputation or disarticulation	227			
Toes	44			
Upper extremity, major amputation or disarticulation	54			
Fingers or thumb	69			
Revision, etc. amputation stump	15			
Operations on fractures	2,739	2,069	670	2,728
Closed reduction without traction	1,819			
Closed reduction with traction	152			
Open reduction without fixation	22			
Open reduction with fixation	64			
Débridement, compound fracture	682			
Other operations on musculoskeletal system	190	105	85	182
Ostectomy and other operations on bones	66			
Arthrotomy and other operations on joints, cartilages or bursa	93			
Operations on muscles, tendons, tendon sheaths and fascia	31			
Operations on circulatory system	75	50	25	72
Skin graft operations	235	180	55	233
Skin graft, split	211			
Skin graft, other	24			
Exploration, débridement, or suture of wound (including removal of foreign body)	1,281	1,200	81	1,276
Of head or neck	129			
Of trunk	208			
Of extremities	944			
Other general or specialized surgery (including blood or plasma transfusion)	616	509	107	607

[1]Includes operations performed for 1st diagnosis only, for 1st and 2nd diagnosis on same patient, and for 2nd diagnosis only.

trast to the 5.5 to 6.0 ratio existent during World War II. Reference to previous publications (1, 2) will indicate the present status of the physician in the Army and the progressive attitude on the part of the Office of the Surgeon General.

The Army was not overwhelmed by returning physician-veterans at the outset of the conflict. The vast majority of the physicians in the country with war experience returned to civilian practice at the conclusion of World War II. Without them, therefore, it was necessary that those experienced men still in service not only overseas but also within our hospital installations for specialized treatment in the continental United States be utilized as efficiently as possible. The influx of new medical officers in the main has resulted in ample numbers, but in a preponderance of men with incomplete or no war-surgery experience, mature judgment, or complete training. It has been necessary that the work load fall on the younger and less experienced, while the older men have been used for supervision, consultation, and guidance. These young men have in a remarkably short time broadened their knowledge, increased their worth, and compiled an excellent record. During the period of study they treated 26,598 battle casualties, of whom only 658 died, a case fatality rate of 2.5 per cent.

Apart from the hundreds of young men not yet accredited in a specialty, Table VII indicates the numbers of those presently on duty in the Far East who are certified or qualified in their respective specialty, and upon whom has fallen the great burden of maintaining accepted surgical standards, supervision, and teaching. It is, of course, our desire that these men be augmented by others equally well qualified. It should be pointed out that in spite of the rigors of warfare, the majority of the hospitals in Japan have been able to conduct teaching programs, conferences, and research. The civilian consultants who regularly visit the Far East assist materially in bringing pertinent information and advice to these hospitals. Indeed the reports of these consultants attest to the success of the program, the intellectual interests of personnel, and adequacy of facilities in the theater.

Since the overall personnel situation has been considerably improved during the past 6 months, it is contemplated that the number of well qualified and experienced surgeons can be increased. There is a rotation policy now in existence which will, of course, influence the numbers and distribution of medical officers. The great assistance which was made possible from the temporary loan of several hundred Navy medical officers cannot be ignored or underrated. These officers materially reduced the personnel deficiency during the early months of the conflict, but even with them on duty it was not possible to staff fully all of the combat units and field installations.

FACILITIES

It was pointed out previously that a well organized hospital base in Japan was available at the outset. This system had been in operation for a number of years, was well organized, and was able to expand rapidly in the face of increased demand. Although greatly overburdened during the fall of 1950, they have since been augmented by other hospitals, including 2 convalescent centers, sent out from the United States. At the present time adequate bed spaces exist for the treatment of all types of casualties, and it has been possible to revert to the usual 120 day evacuation policy. This in turn has relieved the great strain that was placed upon the hospitals in the continental United States which, as is well known, were sharply reduced by economic demands shortly prior to the Korean incident.

Forward hospital facilities have been provided through the use of mobile units including the Army surgical hospitals, field hospitals, and evacuation hospitals. Special teams of surgeons from other Far East hospitals have been attached to these units from time to time in order to provide definitive surgery well forward at the division clearing station level. The flow of evacuees from battalion aid stations and other forward divisional units to the various hospitals has been constantly hampered by virtue of the Korean terrain. Rarely in the past, have American troops been forced to fight under such difficult circumstances.

As a matter of interest, to demonstrate the amount of work done in a forward field unit, the following data were extracted from a report for the period July 9, 1950 through December 31, 1950. This unit, a mobile Army surgical hospital, is typical of all other similar surgical units, and the various other mobile hospitals. When one considers these figures from our field units it is apparent that the case load subsequently evacuated to the fixed hospitals in Japan and the United States was very great.

TABLE VI.—EVACUEES RECEIVED AT HOSPITALS OF DEFINITIVE TREATMENT IN THE UNITED STATES BY PRIOR OPERATION OVERSEAS AND MONTH OF ADMISSION TO HOSPITAL OF DEFINITIVE TREATMENT (SEPTEMBER 2, 1950 TO APRIL 30, 1951)[1]

Month of admission to hospital of definitive treatment	Total No. of cases	Cases without operation	Cases with operation	Per cent with operation
Total Sept. 1950–Apr. 1951	16,711	10,706	6,005	35.93
Sept.	2,473	1,520	953	38.54
Oct.	3,678	2,569	1,109	30.15
Nov.	1,203	773	430	35.74
Dec.	2,644	1,894	750	28.37
Jan.	1,719	1,190	529	30.77
Feb.	1,638	988	650	39.68
Mar.	1,797	969	828	46.08
Apr.	1,559	803	756	48.49

[1]There is an average time lag of approximately 4 weeks from the date on which a patient is first admitted overseas to date admitted to a hospital of definitive treatment in the United States.

Total admissions July 9, 1950 to December 31, 1950.		8,577
Total number of patients operated upon		1,516
Total number of surgical procedures		2,280
Laparotomies	221	
Thoractomies	60	
Amputations	59	
Débridements	1,173	
Head cases	10	
Casts applied	588	
Total deaths for period		112
U.S. Forces	83	
Other	19	
Total number of patients x-rayed		3,732
Total number of x-ray exposures		5,598
Total number of pints whole blood used		1,298

Throughout most of the period noted, this hospital and case load were handled by 10 medical officers, 12 nurses, and the administrative complement of 2 medical service officers, 1 warrant officer, and 95 enlisted men. Just prior to the end of the year, the staff was increased to 17 medical officers, 17 nurses, and

TABLE VII.—NUMBERS OF MEDICAL OFFICERS CERTIFIED OR QUALIFIED IN A SURGICAL SPECIALTY, FAR EAST, FOR PERIOD JULY 1950 THROUGH FEBRUARY 1951

Specialty	Board certified	Board qualified
Obstetrics and gynecology	0	5
Urology	0	3
Anesthesiology	0	15
Ophthalmology	1	4
Otolaryngology	1	5
Neurological surgery	1	0
General surgery	8	37
Thoracic surgery	1	1
Plastic surgery	0	2
Orthopedic surgery	4	10

121 enlisted men. When it is realized that this unit is typical of the work and staffing of the Far East installations, it is apparent that a great work has been accomplished.

EVACUATION

The evacuation of the wounded in Korea has been complicated by the rugged topography and lack of first class roads, both highway and rail. These circumstances have hampered the use of ambulances or converted carriers. Air evacuation in forward areas by helicopter has therefore been carried out to an unusual degree and with outstanding success. From the forward mobile hospitals, as well as the field, the patients are evacuated quickly and easily to other installations, and thence by air to hospitals in Japan. Liberal use of the triad comprised of an air strip, mobile hospital, and division clearing station has been made in order to facilitate these evacuation considerations.

TABLE VIII.—ARMY BATTLE CASUALTY EVACUEES RECEIVED AT HOSPITALS OF DEFINITIVE TREATMENT IN THE UNITED STATES: CUMULATIVE NUMBERS AND PERCENTAGE DISTRIBUTION BY ELAPSED TIME FROM WOUNDING TO FIRST MEDICAL CARE (SEPTEMBER 2, 1950 TO FEBRUARY 28, 1951)

Time elapsed in hours	Cumulative Number	Cumulative Per cent
0 up to ½[2]	980	24.0
½ up to 1	1580	38.6
1 up to 2	2322	56.8
2 up to 3	2755	67.3
3 up to 4	3030	74.1
4 up to 5	3212	78.5
5 up to 6	3359	82.1
6 up to 7	3445	84.2
7 up to 8	3514	85.9
8 up to 12	3694	90.0
12 up to 18	3832	93.7
18 up to 24	3885	95.0
24 and over	4091[1]	100.0

[1]Includes only cases with requisite time intervals specified on Evacuee Report Cards (MED-75) received in indicated period.
[2]Up to but not including.

TRANSPORTATION

The method of evacuation from Korea and Japan of 29,922 casualties is presented from an evacuation report submitted during February 1951 and covers a period of several months prior to that time:

	No.	Per cent
Air evacuation—ambulance cases	13,469	45
litter cases	10,392	34.7
Ship evacuation—ambulance cases	4,878	16.3
litter cases	1,183	3.9

The problems inherent in any rapid evacuation program as they apply not only to the best interests of the wounded and surgical management but also to the maintenance of a fighting force are well recognized, and steps have been taken to ensure that proper consideration is exercised along the entire line of evacuation. While the availability of air evacuation must not alter correct application of surgical principles in order to avoid increase in wound infection, deformity, morbidity, and noneffective rate, speed in forward evacuation and in preparation for definitive surgery is a different matter and is always urgent. A reduction in the interval between time of wounding and treatment will mean the saving of life, saving of extremities, minimizing of deformity, and shortening of convalescence. Furthermore, such action serves to avoid undue dissipation of a fighting force by excessive loss of manpower through delayed transportation and medical care. The time interval from wounding to first admission to hospital was reduced from 2.9 days in July 1950 to 1.4 days in February 1951, averaging 2.4 days for the entire time period. Tables VIII and IX illustrate the elapsed time from wounding to the first medical care and from wounding to hospitalization, respectively, in a large group of casualties subsequently evacuated to the United States. It will be noted that 95 per cent of all the casualties received some type of

treatment within 24 hours from wounding and that 24 per cent received some type of treatment in less than one-half hour. Considering the natural terrain hazards in Korea, the results have been most outstanding and worthy of special emphasis.

The patients were further studied to show type of injury sustained in relation to elapsed time from wounding to treatment. The early locating and transporting of these casualties by medical air men and others go far toward explaining the low mortality thus far attained in Korea.

Detailed study of Table IX will reveal that in a number of cases, the elapsed time from wounding to first hospitalization is excessive. After personal interviews with many of these wounded soldiers to ascertain what factors produced this delay, one or more of the following circumstances existed: capture by the enemy, isolation of the soldier or of a unit, an unfavorable and unstable tactical situation, or terrain barriers to evacuation. In some instances the wounded were initially treated in clearing stations functioning as a forward hospital, then evacuated; therefore the record of first hospitalization occurred on admission to a fixed hospital.

TABLE IX.—ARMY BATTLE CASUALTY EVACUEES RECEIVED AT HOSPITALS OF DEFINITIVE TREATMENT IN THE UNITED STATES: CUMULATIVE NUMBERS AND PERCENTAGE DISTRIBUTION BY DAY FIRST HOSPITALIZED AS MEASURED FROM DAY ON WHICH WOUNDED (SEPTEMBER 2, 1950 TO FEBRUARY 28, 1951)

Day first hospitalized expressed in terms of calendar day on which wounded	Cumulative Number	Cumulative Per cent
Same calendar day (i.e., 0 days following day wounded)	1,973	34.4
1	3,935	68.7
2	4,637	80.9
3	4,993	87.1
4	5,268	91.9
5	5,451	95.1
6	5,524	96.4
7	5,561	97.0
8 or more	5,731[1]	100.0

[1]Includes only cases with requisite time intervals specified on Evacuee Report Cards (MED-75) received in indicated period.

THE PERMANENTLY DISABLED

The total of permanently disabled soldiers for the period under study has been determined in part. Of the 11,638 Army evacuees from Korea, 525 (or 4.4 per cent) presented some type of known permanent disability. This figure, of course, is not the final one for the conflict, but it will serve to indicate relative incidence thus far. These cases are summarized in Table X.

The amputee, hemiparaplegic or paraplegic, the blind, and the deafened are evacuated in the shortest possible time to specialized treatment centers located within regional areas of the United States, so that the patient may have the additional benefit of being hospitalized as near to his home as is possible. Especially trained physicians, nurses, physiotherapists, and corpsmen care for these soldiers. Every effort is made to rehabilitate them into useful individuals who will not be a total charge either to their respective families or to a hospital facility. Thus far, the program has progressed splendidly, results have been outstanding, and the morale of the patients has been high. The Army hospital staffs have worked with interest, enthusiasm, and good judgment. Overall present policy now requires that these cases be transferred to Veterans Administration facilities at an early date, well prior, in many instances, to complete healing of all wounds and complications. It has been thought that such transfers will provide the military hospital with more fluid bed

TABLE X.—ARMY EVACUEES RECEIVED AT HOSPITALS OF DEFINITIVE TREATMENT IN THE UNITED STATES BY SELECTED SPECIFIC TYPES OF PHYSICAL DISABILITY (SEPTEMBER 2, 1950 TO FEBRUARY 28, 1951)

Total No. evacuees from Japan-Korea[1] 11,638
Number with none of the selected disabilities 11,121
Number with selected types of disability ..... 525[2]

Type of disability	Total No.	Type of disability	Total No.
Amputations	200	Hemiplegia	13
Upper extremity one (41) both (1)	42	Paraplegia	69
		Blind one eye (182) both eyes (22)	204
Lower extremity one (140) both (18)	158		
		Deaf one ear (13) both ears (14) one ear with defective hearing in other (12)	39
Upper and lower (any combination)	3[3]		

[1]Includes battle and nonbattle cases.
[2]8 cases had 2 disabilities, making 517 disabled individuals.
[3]Not an addition to amputations; number with combined loss shown also in above appropriate categories.

TABLE XI.—ARMY EVACUEES RECEIVED AT HOSPITALS OF DEFINITIVE TREATMENT IN UNITED STATES BY CAUSATIVE AGENT (SEPTEMBER 2, 1950 TO FEBRUARY 28, 1951)

Causative agent	Number of cases	Percentage
Total—All cases*	7,039	100.0
Small arms weapons	3,102	44.1
Explosive and fragmentation weapons	3,223	45.8
Parachute jump	11	0.2
Land transportation vehicle	120	1.7
Incendiary or other chemical warfare agent	9	0.1
Direct/indirect intended effects of instruments of war	242	3.4
Use of own instruments of war	235	3.3
Falls or jumps on land	61	0.9
Machinery tools, objects, etc.	19	0.3
Miscellaneous, other and unspecified causes	17	0.2

*The infantry constituted 5,082 cases, or 72.2% of the total.

spaces and not cause them gradually to become hospitals for chronic patients. However, as is evident the Veterans Administration hospital must be prepared to receive these patients earlier than ever before, in a state of incomplete convalescence and rehabilitation, and with definitive care still in progress.

CAUSATIVE AGENTS AND TYPE OF WOUNDS

In general our combat casualties have resulted from small arms fire, hand grenades, mortar shells, and artillery. The opposing forces have not used heavy concentrations of artillery, aerial bombing, flame-throwers, and other devices of destruction. Their use of land mines has been on a lesser scale than that encountered in World War II. This fact has accounted in the main for the type of casualty received, the relatively low incidence of burns, total disabilities, chest injuries, and amputations as compared with any given period in World War II. It also accounts in great measure for the high return to duty within the overseas theater. Table XI illustrates the causative agents inflicting injury to 7,039 soldiers who were evacuated to the United States for further treatment. These soldiers represent a cross section of the severely wounded from such agents, and it must be realized that 65.3 per cent of the total casualties during the period used were returned to duty without evacuation to the United States. It is interesting to note that of the 7,039 cases, 72.2 per cent were from infantry units.

It should be pointed out that all of these casualties were not sustained at the hands of the enemy. As noted, many occurred with or from our own troops, air cover, movement hazards, parachute jumps, and road accidents.

In order to simplify the material and still obtain a comparison of type of wound with causative agent, Table XII is based upon the broad categories of injury. It is evident that the use of small arms and hand grenades has resulted in emphasis being placed on trunk and extremity injuries, but with a relative low rate of death and permanent disability as noted previously.

DELAYED CLOSURE OF WOUNDS

By virtue of the contaminated nature of wounds in ground troops, the Army Medical Service must and does advocate delayed closure of wounds. This lesson was well learned in World War II, but it is recognized that many surgeons, especially those without prior war surgery, have a great desire to perform primary suture of wounds. When this occurs, the incidence of infection, wound dehiscence, morbidity, and mortality rise. By constant repetition of the principles of delayed closure, we have been able to reduce complications and hasten healing. It is known that successful primary suture has been practiced in selected cases by some surgeons of wide civilian and military experience, especially on Air Force and Navy casualties. However, such a program cannot be advocated by the Army, whose surgeons must treat from the outset wounds that are grossly contaminated. Medical officers are instructed to close the deeper structures and to leave unsutured the skin and superficial layers. The wound edges are to be held open by the smallest amount of dry, fine mesh gauze. It is recommended that delayed suture be performed at the earliest time, and whenever possible within 5 to 7 days.

During the fall and winter of 1950 many patients had to be evacuated to the United States prior to delayed closure of wounds because of the shortage of bed space. At the present time most secondary closures are done

in the overseas hospitals. It is worth while here to note that fracture cases have occasioned some difficulty because of acute bed shortage in the past. The policy now is to treat definitively and hold these patients in proper fixation for sufficient time in the Far East so that stabilization has occurred prior to evacuation. If this is not feasible, such patients have high air priority and are evacuated at once in temporary fixation, so that they may be treated in a specialized hospital in the continental United States without further necessity for evacuation.

TABLE XII.—ARMY BATTLE CASUALTY EVACUEES RECEIVED AT HOSPITALS OF DEFINITIVE TREATMENT BY BROAD CATEGORIES OF TRAUMATISM AND BY ANATOMICAL LOCATION (SEPTEMBER 2, 1950 TO FEBRUARY 28, 1951)[1]

Categories of traumatism	Number	Per cent	Anatomical location	Number	Per cent
Total cases	7,041	100.0	Total	7,041	100.0
Fracture, compound	3,173	45.0	Head, neck, face	826	11.7
Fracture, other	393	5.6	Trunk abdominal and thoracic	1,124	16.0
Wounds	2,878	40.9	Upper extremity	2,239	31.8
Amputation, traumatic	108	1.5	Lower extremity	2,822	40.1
Burns	26	0.4	General or not specified	30	0.4
Concussion	43	0.6			
Other or not specified	20	6.0			

[1]The categories of traumatisms and anatomical locations are based on the cause of evacuation and do not reflect additional diagnoses or locations.

BURNS

There has been a remarkably low incidence of major burn cases from the war zone. During the period under study, only 26 cases were evacuated to the States primarily for this reason. All other cases were of such degree that complete treatment and return to duty could be carried out within the theater or, if evacuated, were evacuated because of other more serious conditions. Progress in the treatment of burns has been reflected in the liberal use of whole blood transfusions, control of electrolytic balance, early excision of third degree eschars to facilitate skin grafting, the use of chemotherapeutic agents, use of the closed method of treatment, and, more recently, investigations relative to the use and feasibility of the exposure method. It is the Army's policy that all burns should be gently débrided at the first point of definitive treatment, and that smooth, firm pressure dressings be applied, fine dry mesh gauze being used adjacent to the wound. Although some difficulty has been experienced in preventing too frequent inspection of these wounds during evacuation, it has been our policy to leave these dressings undisturbed for 10 to 14 days, utilizing proper immobilization when applicable in the position of function. At the time of change of dressings, steps are taken to prepare the third degree areas for early skin grafting.

Use of the exposure method of treatment has been found to be practicable within 4 to 6 days after injury but has failed completely when used after that period. The method is not altogether suitable to field conditions since it demands that the patient reach a fixed installation within 5 days. Even though this period exceeds the average time for casualty evacuation in Korea, it still implies that such a method cannot yet replace the closed method of treatment in war surgery.

It is probable that the type of warfare waged by the enemy has been largely responsible for the low incidence of major burns. Reference to Table XI will show the predominance of small arms and mortar injuries and the absence of air and armored warfare on the part of North Korean forces. Recently a special burn team visited the Far East and inspected all patients and burn programs. This group reaffirmed the use of current methods of treatment. Investigations with ACTH and cortisone, as with most other reported studies, have not demonstrated any directly beneficial effect on the burns.

COLD INJURY

The high incidence of cold injury (frostbite, trench foot, and immersion foot) among United States Army troops participating in the extensive winter combat operations in Korea has been one of the most important medical problems encountered. The Army Medical Service has been aware of the seriousness of this condition, for in World War II the total record of injuries in the American Army

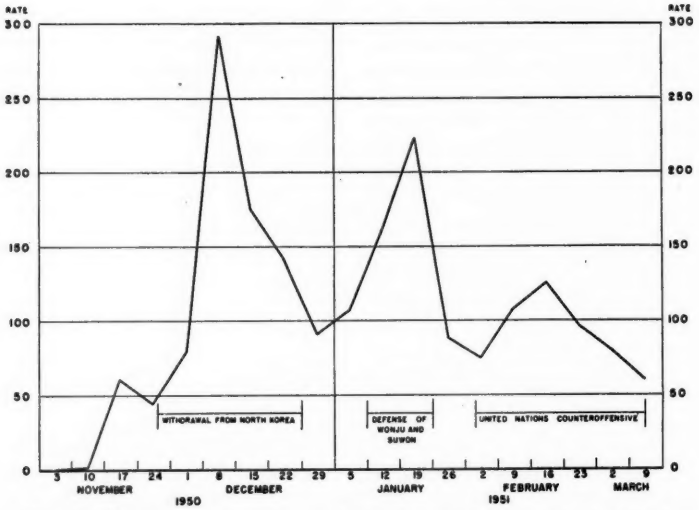

Fig. 1. Incidence (rate in cases per thousand strength per year) of cold injury among United States Army troops in Korea for the period from the week ending November 3, 1950 through March 9, 1951.

due to cold, ground type, was 55,331. The average time away from duty was 55 days; in other words, this figure represents the total loss of a 15,000 man division for 6 months.

The Quartermaster Corps and the Army Medical Service have carried out extensive research in the development of protective clothing, footgear, and training doctrine, in order to permit our military personnel to compete on more even terms with other forces more accustomed to extremes of wet and cold. Research has been conducted at the Army Medical Service Field Research Laboratory at Fort Knox, Kentucky, the Quartermaster Climatic Research Laboratory in Massachusetts, and in field studies and trials in Alaska, Canada, the Aleutian Islands, and in the colder areas of the United States. The development of protective clothing and equipment by these means is believed to have produced an effective group of items. However, it is apparent that such protection alone cannot solve the entire problem. There still must be timely issue of this materiel to troops, acceptance and understanding by the troops, and their proper wearing and maintenance. Indoctrination must not only be offered repeatedly but be emphasized properly to ensure that troops understand and accept the basic importance of personal care and precaution. The natural hazards of extremely low temperatures and fluctuations of temperatures in the incidence of cold injury are factors over which we cannot exercise control. It is equally apparent that the wounded and

TABLE XIII.—SURVEY OF COLD INJURIES RELATIVE TO AMPUTATIONS IN ARMY EVACUEES TO FEBRUARY 21, 1951

	Total No.	Per cent
Cold injuries received in United States from Far East	630	100.0
Cold injuries with amputations in overseas hospitals:		
Major amputations	8	1.3
Minor amputations	5	0.8
Cold injuries with amputations in United States hospitals:		
Major amputations	17	2.7
Minor amputations	57	9.0
Cold injuries returned to duty from United States hospitals	50	8.0
Estimated number of patients presently hospitalized who will be returned to duty, no amputation	381	60.5
Estimated number of patients who will be separated or retired from service for disability, with or without amputation, and pending	112	17.7
Amputations in overseas hospitals		2.06
Amputations in United States hospitals		11.74
Amputations, all areas		13.8
Estimated return to duty		68.5

incapacitated soldier is more prone to cold injury since he is unable to utilize his knowledge and take the necessary preventive measures.

The cold injuries which occurred were due primarily to the very low ambient temperatures together with the extremely difficult combatant conditions and terrain. The factors of poor command function, carelessness, inadequate clothing, or willful intention did not play important roles in the development of frostbite. Furthermore, approximately 35 per cent of these casualties had incurred combat wounds prior to developing frostbite. While we prefer the use of the general term "cold injury," nevertheless the usage of trench foot, immersion foot, and frostbite is so fixed in our terminology and thinking that we still use it for purposes of comparison herein. It has been found during the period September 1950 to February 1951 that approximately 85 per cent of the cases were frostbite and 15 per cent immersion foot. The presence of the rice paddies, heavy rains, and stagnant water in Korea may result in a sharp increase in immersion foot during warmer weather in the spring. The rather free interchange of these terms by personnel in reference to any given case has also occasioned some element of confusion in the compilation of statistics.

In spite of the more severe climatic and tactical conditions, the troops in Korea present a lower incidence of cold injury rate than some of the United States armies in Europe in the winter of 1944–1945. The troops in Korea were far better equipped and indoctrinated in cold weather warfare, and the requirements for such a campaign had been anticipated well in advance. Personal interviews with many of the casualties by the authors proved to us that proper measures were in fact taken.

In our data for September 1950 to February 2, 1951, a total of 393 cold injury Army casualties were evacuated to the United States from Korea. More recent figures for Army personnel show that over 600 have been admitted to zone of interior hospitals for treatment. Table XIII contains data relative to partial or complete amputations which have resulted in these casualties. Approximately 125 of them are currently undergoing plastic hand surgery in order to restore a great measure of function, and thus prevent them from becoming totally disabled. We have advocated ultraconservatism in the surgical sacrifice of tissues subjected to cold injury.

The incidence of cold injury sustained by troops has been to a large extent unavoidable since such injuries have been suffered either in combat operations or after battle injuries in subzero temperatures.

TABLE XIV.—PRELIMINARY REPORT ON MEDICAL ASPECTS OF THE KOREAN CAMPAIGN ADMISSION AND INCIDENCE RATES FOR PERIOD JULY 1, 1950 TO FEBRUARY 23, 1951

Cause of admission	Annual rate per 1,000[1]
All causes	1,561
Disease	905
Nonbattle injury	272
Battle casualty	384
Incidence	**Annual rate per 1,000**
Selected conditions:	
Neuropsychiatric conditions	74.58
Fever of undetermined origin	45.76
Malaria	8.08
Dengue	0.28
Diarrhea and dysentery	46.92
Common diarrhea	36.18
Unclassified dysentery	9.03
Bacillary dysentery	1.07
Amebic dysentery	0.64
Bacterial food poisoning	1.36
Typhoid fever	0.01
Common respiratory infections and influenza	115.70
Pneumonia, all types	17.26
Tuberculosis, all types	1.03
Diphtheria	0.07
Measles	1.13
German measles	0.28
Chicken-pox	0.39
Meningitis, meningococcic	0.23
Mumps	0.52
Rheumatic fever	0.71
Streptococcal sore throat	0.80
Venereal disease	57.96
Gonorrhea	32.73
Syphilis	3.49
Other	21.74
Scabies	1.58
Encephalitis	6.41
Infectious	3.77
Acute, unclassified	2.64
Encephalomyelitis	0.15
Hepatitis, infectious	21.93
Mycotic dermatoses	4.25
Poliomyelitis, acute anterior	0.53
Gas gangrene	0.24
Relapsing fever	0.13
Mononucleosis, infectious	0.09
Small pox	0.09

[1]Rate expressed as admissions or cases per 1,000 mean strength per annum.

Figure 1 demonstrates that the incidence of cold injury in Korea has been greatest during periods of heavy combat in the cold season.

DISEASE

Disease always takes its toll of a people, whether military or civilian. Emphasis in the Armed Forces must, therefore, necessarily be focused on prevention and reduction, in order not to render a combat force ineffective by virtue of epidemics. While this is primarily a presentation for the surgeon, nevertheless the data on disease are of sufficient interest to be included. Work in preventive medicine has been of recognized importance for many years, and, when a conflict is waged in such unfavorable territory as that of the Korean peninsula, efforts must be redoubled. The virtual nonexistence of tetanus as a complication of war wounds in United States troops must not be slighted when one reviews preventive medicine as a whole. As is well known to most people of military experience, definite prophylactic measures through active immunization are taken against small pox, typhoid, paratyphoid, typhus, and cholera.

Table XIV illustrates the incidence of various illnesses found in our troops during the period of study. Absolute numbers cannot be given, but rates expressed as admissions or cases per 1,000 strength per annum are given so that comparisons of incidence can be made. In only 2 months during the period shown have nonbattle and battle casualties exceeded the numbers incapacitated by disease.

It is known that the incidence of disease in enemy troops in Korea far exceeds the figures noted, and in some cases the incidence of such diseases as small pox and typhus fever has been in epidemic form. It will be noted that infectious hepatitis ranks well up on the list of frequent diseases. Special work is being done overseas at this time to learn more about the cause, prevention, and therapy.

USE OF CHEMOTHERAPEUTIC AGENTS

Penicillin, streptomycin, aureomycin, terramycin, and the sulfonamides have been available and are used in the Korean conflict. It has been emphasized that these agents will not be used locally in wounds and that indiscriminate administration will not be indulged in after the initial early treatment. All wounded receive penicillin and/or sulfadiazine from the onset of medical aid and during the acute phases of treatment. In the event of chronicity, severe infections, doubtful infections, infections not responding to treatment, all staphylococcal infections, and nonhemolytic infections, culture sensitivity studies are made in order to utilize the specific agent of choice. Systemic chemotherapy, however, is emphasized as an adjuvant to correct surgical management of wounds, clean, contaminated or infected. We teach that their use does not offset the hazard of devitalized tissue, dead space, inadequate or improper drainage, nor substitute for accepted surgical principles. The low morbidity and mortality thus far have reflected a judicious combination of proper surgical approach and use of the chemotherapeutic agents. The Army Surgical Research Unit conducts constant studies along these lines.

COMMENT

An early review of the results in Korea has been presented, viewed especially from the surgical standpoint, with some comment on disease. While this report of present status reflects primarily the work done in a 6 months' period of observation on a group of evacuees, however, it does appear timely and appropriate that this much of the story be presented. The results thus far have been outstanding and are a creditable record of performance. One must give full credit to the sound lessons from World War II that have been applied so early in this conflict, to the work and efforts of the medical officers and other personnel in the Far East, to the cooperation and visits of civilian consultants to the theater, to the early forward availability of whole blood and antibiotics, and to the advantages of air evacuation. The low mortality and morbidity and the high percentage of soldiers returned to duty reflect high credit upon all concerned.

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A STANDARD CONTACT BURN

Method of Production and Observations on the Blood Picture Following its Production in Dogs

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MEDICAL literature contains many articles dealing with studies made on experimental burns. A variety of laboratory animals have been used including dogs, rabbits, guinea pigs, mice, rats, and pigs. Most investigators have devised their own methods of burning and great varieties of surface areas and depth of burns have resulted. Active flame, steam, water, and burning irons have been employed for burn production (1, 5, 6, 11). A surprising number of studies have been done on animals burned so intensely as to cause early death; such experiments were usually conducted to study the responses to the most severe burns.

We have accumulated data following the production of a burn which we consider to be an accurate "standard burn." In developing the standard burn we were influenced by the following important considerations which should be fulfilled:

1. The burn should involve a constant percentage of the body surface area.
2. The burn should involve a constant depth of destruction of the skin thickness.
3. The burn must be easily and accurately reproducible from animal to animal and from laboratory to laboratory.
4. The burn should be only of such extent and severity as to allow complete healing without therapy and should be one in which the degree of infection will not be cause for major consideration. (This point could be omitted for studies involving burns of severe depth and percentage area which entail short survival periods for the animal.)
5. The burns should not involve the legs, in order that freedom of movement and good veins for venipuncture are maintained. Also body orifices and surrounding areas must remain unaffected so as not to interfere with eating, voiding, and defecation.
6. The laboratory animal used should be one easily handled and maintained under control conditions.

By securing control data on such a burn, we have gained a most valuable tool by which variants, both beneficial and detrimental in nature, could be accurately evaluated.

This paper is devoted to a detailed description of the method of production of a standard burn with observations on the blood picture following its production.

LABORATORY ANIMAL

Young and middle aged (2 to 4 years) mongrel dogs, male (13) and female (25), were used. They were housed in regular kennel cages in rooms without temperature control or in outside areas for exercise, and were fed a standard diet of dog chow pellets only, water being given as desired. Each dog was kept in the kennels for a period of between 2 and 4 weeks before burning (average 3 weeks) during which time it received a vermifuge for removal of intestinal parasites. Any skin abnormality such as mange, or signs of distemper, such as sneezing, running nose, watering red eyes, diarrhea, or persistent elevation of rectal temperature, were critera for discarding a dog. Female dogs (a total of 6 in this series) in whom there were any signs of possible pregnancy were operated upon, and a supracervical hysterectomy was performed, followed by adequate postoperative recuperation. Fol-

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lowing this control period all dogs were considered in a "normal state of health" when peripheral blood studies were at normal levels.

SKIN SURFACE AREA

Most investigators have derived formulas which relate surface area as a factor of body weight. The simplest conception of this relationship is that solids of similar shape and the same density will have surface areas equivalent to the 2/3 power of their volumes or weight. It is apparent, however, that animals of greatly different configuration, such as dogs, would in all probability not have surface areas represented by this simple 2/3 relationship alone. A formula relating the surface to volume, namely, surface = K × volume, 2/3 was developed by Meeh (1879) and later formulated into the "surface area law" by Rubner (1883). If specific gravity is assumed to be constant, weight may be substituted for volume in the expression. The value of K in the above formula was found to vary with different species depending probably upon the pattern shape for the species. The Meeh-Rubner formula expresses surface area purely as an exponential function of weight (3).

We believe the formula most suitable and accurate for our studies is the one devised by Cowgill and Drabkin. The Cowgill-Drabkin formula:

$$A = K \times W^{\bullet 7} \times K_2 \frac{\sqrt[3]{W}}{L}$$

is essentially the Meeh-Rubner expression multiplied by a factor correcting for the nutritive state of the subject.

In this formula:

A = Surface area in square centimeters
K = Constant, equal in dog determination to 6.67
W = Weight of animal expressed in grams
K_2 = A second constant relating to the state of nutrition and equal in dogs to 0.34
L = Length (tip of nose to anus or base of tail) in centimeters

$\frac{\sqrt[3]{W}}{L}$ = the ratio of the cube root of the body weight in grams to the body length in centimeters. (This indicates the nutritive state observed in the individual case in question.)

In our present study we have elected to produce and study burns involving 20 per cent of the total skin surface area as determined by the Cowgill-Drabkin formula.

METHOD FOR BURN PRODUCTION

We have used a modification of the burning iron first proposed by Leach, Peters, and Rossiter. This apparatus is similar in principle to the small glass bulbs used by Hadack and McMaster (1932) for heating mice ears. Our present modification is represented in Figure 1.

Water is circulated through the burning iron from a thermostatically controlled hot water bath at a temperature of 60 degrees C. The bulb of the thermometer in the burning iron rests on the chamber side of the burning surface and its reading is considered that of the burning iron. We were able to keep the temperature quite accurately at 60 degrees C. with variation of only ±0.2 degrees C.

The burning surface is circular, brass metal. The area of the burning surface is 20.8 square centimeters. However, measurements which were made of the area of edema produced following application of the iron at 60 degrees C. for 1 minute showed the average area of edema in many determinations was 23.8 square centimeters and this figure was used as the size of the individual burn when calculating the number of burn applications necessary to produce a 20 per cent surface burn.

Mendelssohn and Rossiter, using the iron described by Leach and associates and a subsutaneous thermocouple which rested at the lower margin of the dermis in guinea pigs, have shown that the average temperature reached at this level during a 1 minute application of 60 degrees C. was 48.4 degrees C. Thus, the tissues affected by the burn are subjected to temperatures ranging from 60.0 to 48.4 degrees C. depending on their distance from the burning iron.

Great care must be exercised in the production of individual burns in order that they may be kept standard. No excessive pressure is exerted on the iron during application, only the weight of the burning apparatus (6.5 pounds when filled with

water). The gripping handles only regulate position and level of the iron and prevent its slipping about on the skin once it is applied. A constant vigilance of the thermometer reading and of timing at 60 seconds is essential to ensure identical burns. Variation in these constants will decidedly affect extent and degree of damaged tissue.

Guided by the work of Leach and associates in guinea pigs, we found that in dogs the burning iron described, held at a temperature of 60 degrees C. for 1 minute's duration, caused necrosis of tissue and a resultant burn comparable in depth to a deep second degree burn in humans. Over bony prominences, such as the hip and scapula, this burn was often third degree in depth.

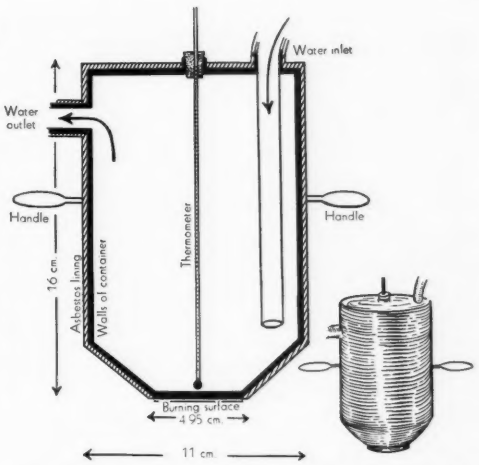


Fig. 1. Diagram of burning iron which was used in these experiments to produce deep second degree burns after application of the iron for 1 minute at a temperature of 60 degrees C.

PREPARATION OF DOG

All dogs are carried through a control period as described previously. On the afternoon before the morning of burning, all hair from the back, thorax, abdomen, flanks, thighs, and upper forelegs is shaved off with an electric clipper. This is followed by careful shaving with soap, water, and razor. Care is taken to produce as little skin abrasion as possible while shaving.

Animals must be shaved no earlier than indicated because hair stubble will grow out in 24 to 48 hours to such an extent as to insulate the skin from effective burning. After shaving, the animal is dried thoroughly, weighed, length measured, and placed in a warm kennel. Food and water are withheld for 12 hours prior to burning.

CHOICE OF ANESTHETIC

Leach and associates have found that neither ether nor sodium pentobarbital at equal levels of anesthesia affect the nature, macroscopically or microscopically, of the burned area.

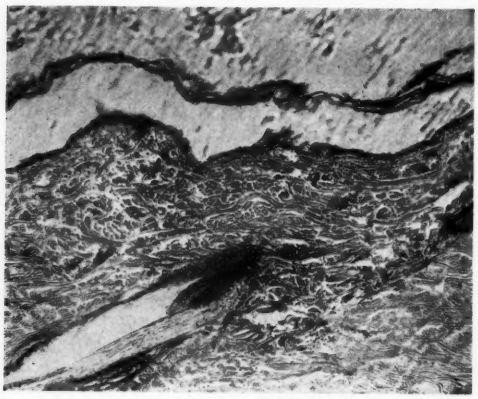
Mendelssohn and Rossiter recommend the use of sodium pentobarbital as the anesthetic in producing burns because they found less fall in body temperature (as well as skin temperature) with this anesthetic than with ether for corresponding levels of anesthesia. This observation is important because, in general, the higher the initial skin temperature the higher will be the temperature reached subcutaneously.

For these reasons and because of the ease of administration and maintenance of adequate levels of anesthesia throughout the burning procedure with a single dose, we have used routinely intravenous sodium pentobarbital as the anesthetic agent.

TECHNIQUE OF BURNING

On the morning of the experiment the dogs, removed from their cages within 15 minutes after awakening and without water and food for 12 hours, were considered "basal." Control blood samples were drawn and blood volume determinations done with T-1824 Evans blue dye. The dogs were then anesthetized with intravenously administered sodium pentobarbital in doses of 30 milligrams per kilogram.

We have found that when burning dogs 20 per cent of their body surface at 60 degrees C. for 1 minute, it is best to place each burn far enough from the adjacent burns so that there is an intervening area of normal skin. This is important for three reasons: first, if individual burns touch or overlap, one cannot be certain of a true 20 per cent



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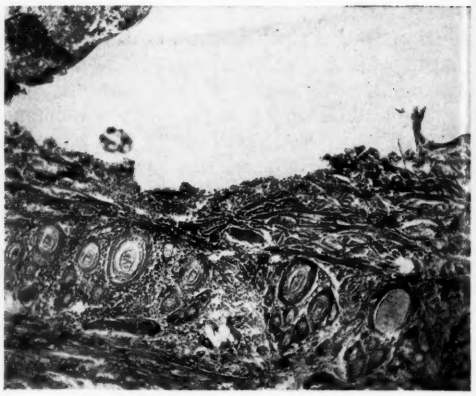

b

Fig. 2. a, Section of dog's skin, taken from the abdomen 45 minutes after burning. There is marked intradermal edema. There is blurring of the epithelium, the cells of which do not take stain well, beginning leucocytic infiltration of the deep tissues, and dilatation of capillaries. b, Section taken from back 2 weeks after burning. There is marked leucocytic infiltration, abscess formation, and beginning epithelial proliferations from margin.

burn; second, when one continuous area is burned the mortality rate is greatly increased due to such large denuded and grossly infected areas; and third, healing is greatly delayed, and at times impossible without therapy, when such large continous skin area are destroyed.

Burning by this method usually takes 40 to 70 minutes (average of 60 minutes), depending, of course, on the number of burns necessary to apply to achieve a 20 per cent surface burn.

A blood sample is drawn for hematology immediately upon completion of the burn and then every 2 hours for 24 hours. During the healing phase blood studies were made every 3 or 4 days until burns were all healed and blood studies showed a return to preburn levels.

STUDIES MADE

Blood samples were drawn from the femoral veins, except the control samples and samples at 2 and 6 hours after burning, when blood volumes were done. These samples were then taken from the jugular veins. Ten cubic centimeters of blood was drawn for each blood volume determination and 3 cubic centimeters for each postburn sample.

Heparin was used as the anticoagulant. Rectal temperatures were recorded at the time each sample was drawn. The weights were recorded with each sample, except those drawn during the first 24 hours following the burns.

The following hematology studies were done on each sample drawn:

1. Hemoglobin—duplicate determinations of oxyhemoglobin by Junior Coleman spectrophotometer (2).
2. Hematocrit—with Wintrobe hematocrit tubes (17).
3. Red blood cell count—single determinations (18).
4. White blood count—single determinations (18).
5. Differential count—Wright's stain for staining (18).
6. Total plasma proteins—by copper sulfate specific gravity method (14).
7. Blood volumes—with T-1824 Evans blue dye. Determinations were made on plasma. One sample was drawn for determinations at 12 minutes after dye injections (13).
   a. Control
   b. Twelve dogs at 2 hours after the burn
   c. Twenty-three at 6 hours after the burn

CONTROL STUDIES

Since the burn production was always accomplished with the experimental animal anesthetized, it was necessary to determine what influence, if any, sodium pentobarbital had on the blood studies which we were interested in observing at the time intervals studied. The sodium pentobarbital was giv-

en intravenously in doses of 30 milligrams per kilogram of body weight.

The dogs used in this control study were handled and managed exactly as those used for burning experiments and blood studies were done at intervals after induction of anesthesia corresponding to the same intervals after burning. Prior to these studies the animals were shaved and food and water were withheld for 12 hours. Immediately before inducing anesthesia with sodium pentobarbital, control samples of blood were drawn and blood volumes determined.

The zero (0) hour sample in burn experiments is the one withdrawn immediately following completion of 20 per cent burn and on an average was taken 1 hour after the induction of anesthesia. Thus, this sample corresponds to the 1 hour postinduction of anesthesia sample that was studied in the control series. The 2 hour sample corresponds with the 3 hour postinduction anesthesia sample, and so forth. Of the total of 40 dogs subjected to this analysis, approximately 50 per cent were later used for the actual burning studies.

During the 24 hour study of each dog a total of 41 cubic centimeters of blood (4.1 per cent of blood volume) was withdrawn for analysis. This was not replaced by other blood or fluid.

Studies were made on 40 dogs 1 hour following induction of anesthesia, and on 20 dogs for the succeeding interval samples. In summarizing the reaction by each dog at each hour studied, we have expressed the change as percentage change, plus or minus, from the control for that dog; for example,

Sample	Hemoglobin grams
Control	15
1 hour after induction of anesthesia	12
Change	−3 (20% decrease)

Each dog's reaction was then averaged with all dogs for each hour to determine the average response.

Level of anesthesia. All dogs were in the third plane of anesthesia at 1 hour. At 3 hours, 5 per cent had reacted, but were unsteady on their feet; 30 per cent had begun to react, that is, were whining and moving their legs slightly; 65 per cent were still soundly asleep. At 5 hours, 80 per cent had reacted and 20 per cent were still sleeping soundly. By the seventh hour 95 per cent had reacted, but only 50 per cent were able to drink water, which was given only after blood volume determinations. At 9 hours all animals had reacted enough to take water by mouth.

Temperature. One hour following the induction of anesthesia in the 40 dogs the rectal temperature had dropped an average of 3.8 degrees F. below control values. It continued to fall, reaching a minimum of 5.4 degrees F. below control values at 3 hours after induction of anesthesia. This is undoubtedly the reason for the marked shivering evident in the dogs at this stage and for 1 to 2 hours hence. At the fifth hour the temperatures had begun to rise and were approximately normal at 9 hours. Twenty-four hours following the experiment they were essentially at control values.

Weight. Only control and 24 hour weights were recorded. At 24 hours the weight had increased an average of 4.2 per cent over the control values. This we interpreted as being due to the consumption of water and food after reacting from anesthesia.

Hemoglobin, hematocrit, red blood cell count. The greatest change in these values occurred 1 hour following induction of anesthesia. At this time, all values had decreased an average of approximately 20.0 per cent below control values. Following this sharp decrease, the values gradually climbed back to normal values by the seventh or ninth hour samples. Twenty-four hours following the experiment the values were slightly below normal, due, in part, to hemodilution following water ingestion.

White blood cell count. The maximum decrease in white blood cell count was 1 hour following the induction of anesthesia when it reached an average of 24.0 per cent below control values. Following this the count gradually returned to normal or exceeded normal by an average of 34.6 per cent at the ninth hour. The following day the count was still high, averaging 38.2 per cent in the 20 dogs studied.

TABLE I.—REPRESENTATIVE VALUES CALCULATED FROM AVERAGE CONTROL DATA* AND AVERAGE PERCENTAGE CHANGE FROM CONTROL AFTER NEMBUTAL ANESTHESIA (30.0 MGM./KILOGRAM BODY WEIGHT)

Time hr. after anesthesia	Wgt. kgm.	Temp. °F.	Hemoglobin gm.	Hematocrit per cent	R.B.C. million/ cu. mm.	W.B.C. No./ cu. mm.	Plasma proteins gm.	Blood volume c.c./kgm.	Plasma volume c.c./kgm.	Red cell mass c.c./kgm.
Control	10.5	101.9	14.1	43.8	6.90	13,900	6.65	95.6	54.4	41.7
1		98.0	11.4	35.2	5.60	10,440	6.12	86.4	55.7	31.1
3		96.4	12.2	37.4	5.75	11,134	6.21			
5		97.5	13.1	40.3	6.22	12,756	6.26			
7		99.4	13.7	41.7	6.43	15,651	6.37	90.4	52.3	38.6
9		100.9	13.7	40.5	6.47	18,709	6.36			
24	11.0	101.2	13.4	39.6	6.35	19,210	6.34			

*Average of 40 dogs at 1 hour and 20 dogs at 3, 5, 7, 9, and 24 hours.

Differential counts. No significant changes or trends were noted in the animals studied except moderate shift to the left with few immature cells in the 9 and 24 hour smears.

Plasma proteins. Total proteins decreased a maximum at the 1 hour sample, an average of 7.9 per cent. This gradually climbed to normal as the seventh and ninth hour samples were reached.

Blood volumes. Determinations of the blood volume at various levels of anesthesia, corresponding in time interval to those studied following burn production, were made. Blood volumes done in 20 dogs an hour following induction of anesthesia showed that at this time there was an average decrease of 25.3 per cent ± 8 in the circulating red cell mass, with only slight increase 2.3 per cent ± 10.8 in plasma volume. This fact indicated that the apparent dilution of the peripheral blood was due mainly to removal of red blood cells from the active circulation at this time and not to an increased fluid content of the circulating blood. This effect of sodium pentobarbital anesthesia has been observed and studied by many workers. Hahn, Bale, and Bonner, Jarcho, and Hausner, Essex, and Mann believe the major portion of red cells removed from active circulation become stagnated in the sinusoids of the spleen. Hausner, Essex, and Mann outlined the spleen with lead pellets and found with x-ray studies that the spleen enlarged remarkably in dogs under sodium pentobarbital anesthesia, the maximum dilatation occurring in 20 to 30 minutes with return to normal size in 6½ hours.

Blood volumes done in 19 dogs 7 hours following the induction of anesthesia showed the plasma volume and blood volume to have returned to approximately control levels, as did the peripheral blood studies.

Mean corpuscular volume. Since the decrease in hematocrit and thus red cell mass as calculated from the T-1824 Evans blue dye method of blood volume determination could be due to a decrease in red cell size, we determined the mean corpuscular volume on each control sample and on each sample taken 1 hour after induction of anesthesia. Average of 40 dogs showed a decrease of 2.3 per cent in mean corpuscular volume values following anesthesia. Thus, change in red cell size probably played little, if any, role in the lower hematocrit value accompanying the anesthetic state.

Table I and Figure 3 summarize our finding in the dogs studied under sodium pentobarbital anesthesia.

EXPERIMENTAL STUDIES

Upon completion of the burning, samples were drawn for peripheral blood studies as described previously. An average of 62 cubic centimeters of blood (6.0 per cent of blood volume) was withdrawn over 24 hour period immediately following burn. No replacement of withdrawn blood. The same system of percentage change from control samples was followed here as was used in the control studies under sodium pentobarbital to arrive at an average response for the 38 dogs studied. Results of the studies on the 38 burned dogs are summarized in Table II and Figure 4.

Therapy. No attempt was made in any way to replace fluid loss, aid nutrition, or combat infection in these dogs. These studies will form our "base line" from which we may later better evaluate fluid, dietary, antibiotic, and chemotherapeutic aids to the healing and recovery from such injury. Also factors which may have detrimental influences on this picture can now be studied with more accurate data on which to base conclusions.

Mortality. Of the 38 dogs which received our standard burn 5 (13.2 per cent) died early (5 to 14 days) in the postburn period. Death was due in 3 instances to bilateral pneumonia with multiple small abscesses throughout both lungs. Two died as a result of generalized sepsis, due in large part to infections of the lower legs. Marked edema of the hind legs and less marked of the forelegs was of quite frequent occurrence.

Level of anesthesia. Eighty-five per cent of the dogs burned had begun to react slightly shortly after the completion of the burning procedure. This was evident by the beginning rise in rectal temperature, whining, and purposeless movements of the extremities. Ninety per cent of the animals had completely reacted 6 hours after completion of the burn. No dog was given water or food until after the 10 hour postburn sample was drawn.

Temperature. At the completion of the burn the rectal temperatures were at their lowest level, an average of 2.3 degrees F. below the control values due in all probability to the effect of the anesthetic. During the 24 hours immediately following the burn the highest temperatures were reached at 6 hours after burn when the average rise was 1.3 degrees F. above control values. During the rest of this period the rectal temperatures remained slightly below the control values. Throughout the healing phase of the burns the highest average level of temperature elevation above control values was 1.1 degrees F., which was maintained from the second day through the tenth day after the burning. This corresponds well with the period of highest white blood cell elevation, shift to left in differential count, and clinical evidence of infection of the burn areas. However, we believe that rectal temperatures in dogs are of very little aid in diagnosis and follow-up because of the great variance in normal values. Excitement from being placed on the treatment table will often cause an elevation which may be quite misleading. Generally, however, acute infectious processes will elevate the rectal temperature to 104.0 degrees F. or above, but in advanced stages of infection the temperature will be found below 100.0 degrees F. Only those animals which died from the burn showed temperatures above 104.0 degrees F. or below 100.0 degrees F. (except, of course, when under sodium pentobarbital anesthesia).

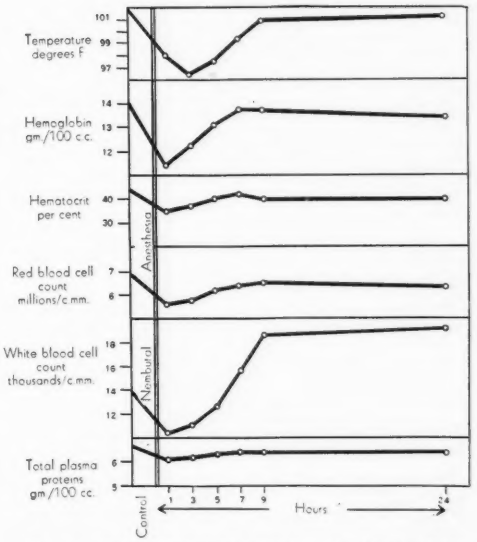

Fig. 3. Composite graph showing the blood picture and temperature changes following intravenous sodium pentobarbital, 30 milligrams per kilogram of body weight. Drawn from figures in Table I.

Weight. The dog's weight decreased gradually reaching the average minimum of 14.4 per cent below control values between 18 and 23 days after burning. The greatest decrease in weight corresponds to the low points in hemoglobin, hematocrit, and red blood cell count values. The burns were clean and not grossly infected and epithelization from the margins was rapidly progressing. Following the low point, these values rise slowly together and epithelization of the wound takes place more rapidly and effectively. In the 38 dogs studied complete wound healing is evident on

TABLE II.—AVERAGE* ABSOLUTE VALUE PER CENT CHANGE FROM NORMAL FOLLOWING 20 PER CENT BODY SURFACE BURN AT 60°C. FOR 1 MINUTE

Time after burning	Wgt. kgm.	Temp. °F.	Hemoglobin gm.	Hematocrit per cent	R.B.C. million/ cu. mm.	W.B.C. No./ cu. mm.	Plasma proteins gm.	Blood volume c.c./kgm.	Plasma volume c.c./kgm.	Red cell mass c.c./kgm.
Control	11.8	102.0	14.9	46.0	6.64	14,500	6.70	95.6	54.4	41.7
Hours 0		99.7	16.9	51.0	7.64	14,645	6.25			
2		101.4	18.3	53.4	8.04	21,605	6.18	86.3	39.8	47.2
4		102.6	18.8	54.0	8.34	27,608	6.16			
6		103.3	18.6	55.8	8.44	31,320	6.22	82.1	38.5	44.5
8		102.9	17.9	52.8	7.94	33,060	6.02			
10		102.2	17.1	51.0	7.54	34,945	5.82			
12		102.1	16.5	48.7	7.14	34,800	5.42			
14		101.4	16.5	49.0	7.34	35,815	5.45			
16		101.4	16.2	47.4	7.14	33,640	5.44			
18		101.6	15.6	46.5	7.04	33,785	5.35			
20		101.7	15.2	45.9	6.87	32,480	5.37			
22		101.7	15.0	45.0	6.70	32,770	5.32			
24	11.84	102.7	14.5	42.5	6.43	29,580	5.42			
Days Group I 2–3	11.4	103.1	12.5	35.6	5.46	27,150	6.23	100.5	60.9	39.5
Group II 6	11.0	103.1	11.9	34.3	5.11	31,460	6.34	96.5	60.2	35.5
Group III 10	10.3	103.1	11.5	34.6	4.89	36,540	6.29			
Group IV 18–23	10.1	102.0	10.7	31.5	4.58	29,300	6.73			
Group V 29–33	10.1	102.6	12.1	34.1	5.13	18,100	6.57			
Group VI 41–46	10.8	101.9	14.2	40.2	5.74	14,270	6.39			
Group VII 54–60	11.2	101.8	14.6	44.0	6.40	14,800	6.50			

*Average of 38 dogs.

an average of 6 weeks after burning, whereas return to control weight is delayed to about 7 or 8 weeks.

Hemoglobin, hematocrit, red blood cell count. The values for all of these increase gradually to an average peak of approximately 20 to 25 per cent above control values at 6 hours after burning. The most dramatic changes occur between the control sample and the completion of the burn and between burn completion and the 4 hour sample. It seems probable that the hemoconcentration in these early hours after burning would be even greater were it not for the tendency of the anesthetic to produce an opposite effect during this period. The change between the 4 hour sample and the 6 hour sample is small and most, if not all, effects of the anesthetic on the blood picture are no longer evident. At the eighth hour sample the drop in these values is definite and continues on down gradually until the sample taken 24 hours following the burn production at which time the values are equal to or only slightly below the preburn control studies. Water was given the dogs following the 10 hour sample, thus this drop from the 10 hour sample to the twelfth hour sample is probably greater than would otherwise be the case.

The most dramatic change in these values indicating anemia occurs on the second and third days after burning when the values drop an average of approximately 13.0 per cent. The decrease is then constant but more gradual until the low point is reached at 18 to 23

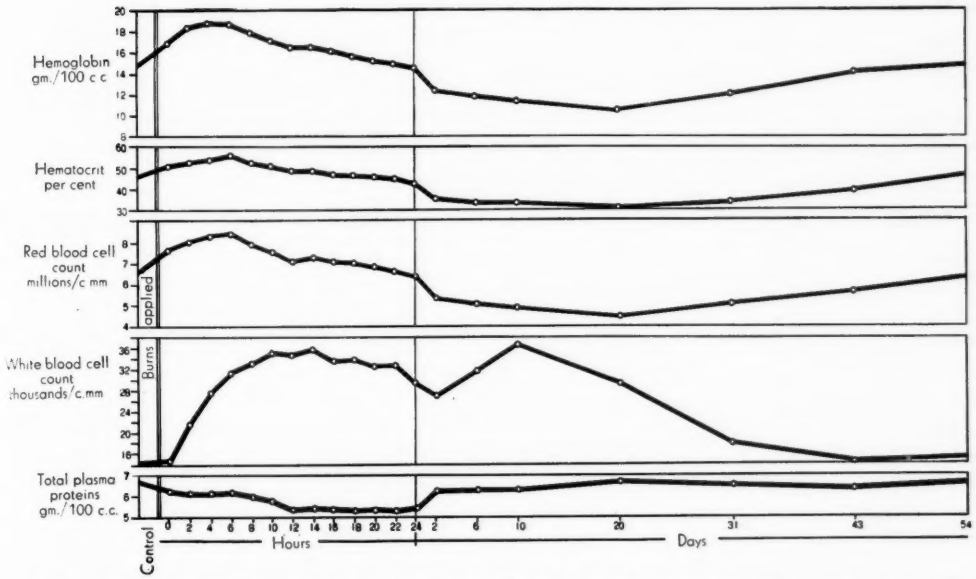

Fig. 4. Composite graph showing the blood picture following a 20 per cent body surface burn at 60 degrees C. for 1 minute. Drawn from figures in Table II.

days after production of burn. The values gradually climb to control levels in an average of 8 weeks. This return to control levels of the blood studies corresponds to the return of the dog's weight to control levels.

White blood cell count. The white blood cell count rises slowly from the beginning of the burn to an average of 2.3 per cent above normal at the end of the burning. Then it rises more rapidly to an average of 147 per cent above control values at 14 hours after burning. This value approaches the peak reached in the period of maximum infection 10 days after burning, averaging 152 per cent above control values. The white cell count remains above normal throughout the postburn period until about the forty-first day when it returns to control levels.

Differential counts. Beginning with the fourth hour after burning the differential count shifts toward the left with early forms of polymorphonuclear leucocytes being evident in increasing numbers. By 12 hours there is a marked shift to the left, usually from 96 to 98 per cent polymorphonuclear leucocytes and from 2 to 4 per cent lymphocytes resulting. This marked shift to the left with numerous early forms of polymorphonuclear leucocytes is maintained through the 18 to 23 day studies, after which there is a gradual return to the control differential.

Plasma proteins. The plasma proteins show a decrease from control values from the very first sample drawn following the burn. This reaches a minimum averaging approximately 20.0 per cent below control values at 12 hours after burning and maintains this low level throughout the remainder of the 24 hour postburn period. Following the first 24 hours after the burn, the plasma proteins are not as depressed as during the period of from 12 to 24 hours, but they do remain below normal values in most cases.

Blood volumes. Blood volumes were studied 2 hours following the production of the burn in 12 dogs. These studies showed that the plasma volume which is not influenced by the anesthetic was decreased an average of 26.9 per cent below the control values. This represented an average loss of 17.0 cubic centimeters of plasma per kilogram of body weight in the 12 dogs.

Blood volumes studied in 23 dogs 6 hours after the burn showed a decrease of plasma volume averaging 29.3 per cent below the control levels. This figure represents an average loss of 18.0 cubic centimeters of plasma per kilogram of body weight and corresponds to the postburn period when the peripheral blood studies indicate the greatest hemoconcentration. It appears from these blood volume determinations that the maximum decrease or loss of fluid from the circulating blood volume to the tissues occurs in the first 2 hours following the completion of burning. This corresponds to the period of visible maximum edema at the local site of the burn. The loss is apparently sustained constantly for a period of at least 6 hours following production of the burn.

It is interesting that work done in this laboratory on standard burn dogs shows similar plasma losses at 6 hours when several blood samples were taken to establish a dye disappearance rate (15).

In our studies, as stated previously, only one 12 minute sample was taken for blood volume determination. Thus, it appears from these present studies that a single sample which is taken 12 minutes subsequent to dye injection is reliable in determining plasma volumes in shock states following burn production.

Blood volumes done at 2 days following the burn show an increase in plasma volume (+12.0 per cent) and a decrease in red cell mass (−5.2 per cent).

This increase in plasma volume is evident at 6 days after burning (+10.7 per cent), and the red cell mass is more decreased (−14.8 per cent).

Autopsy findings. Following the healing of all burn areas, the return of weight and blood studies to control values, each dog was sacrificed with an overdose of sodium pentobarbital. Complete autopsies were performed and tissues from each organ studied. No gross abnormalities were observed at the time of the autopsy. Sections of each organ were studied after staining with hematoxylin and eosin stain. No abnormalities which could be attributed to the burn were found in the sections studied.

DISCUSSION

It is important to remember that this report deals only with the effects of low temperature thermal injury produced by direct contact of the skin with a dry iron at a constant temperature of 60 degrees C. over relatively long (1 minute) periods of application. With this burning iron at 60 degrees C. and a 1 minute application, we have been able to produce repeatedly a series of burns with the same depth of destruction of epidermal tissues. We learned early that great care must be taken in applying the burning iron so that it is in proper contact with the skin.

One obvious objection to this method of producing the burn is the relatively long time necessary to produce the total 20 per cent burn. It is plain that the condition of the dog during the time that the last burns are being made is not the same as when the iron is first applied, because of plasma loss into the burned areas.

However, this does not seem to affect the total picture greatly, if at all, so that the response is essentially the same as clinical cases followed over long periods of time. The plasma loss in these experiments appears to be of the same order of magnitude as in patients suffering the same extent of burn; the details of this comparison will be published later.

It should be mentioned that this method of burn production is applicable only in producing burns up to 30 to 35 per cent of the total skin surface area. Larger surface area burns are not possible because of the need for spacing of burns and animal configurations.

The dog's skin does not compare in every detail with that of humans, but the response to thermal injury is much the same in many respects. The burns which were produced and followed histologically compare to deep second degree burns in humans, except for the lack of typical blister formation. As previously stated, blister formation as it is seen clinically has been observed in our dogs only on the nipples of female dogs. However, local fluid loss into the area of cutaneous thermal injury is most marked macroscopically; and microscopic sections of these tissues show the fluid to be intradermal. Further evidence of this local fluid exodus from the circulating blood

volume has been attained through studies of the plasma volume in burned dogs. These plasma volume studies show loss of large quantities of fluid into the burned tissues at 2 and 6 hours following the completion of the burn. Still further evidence of the fluid loss in these burn experiments is demonstrated by the hemoconcentration curves during the first 24 hours following the burn. Plasma protein levels drop showing protein loss along with fluid loss.

Questions may be raised concerning the 12 hour restriction of water consumption prior to burning and then another 10 to 12 hours' restriction following burns, a total of nearly 24 hours without oral or parenteral fluid. Such prolonged dehydration might cause hemoconcentration without burning. That such is not the case can be seen from the control studies carried out with the animals under sodium pentobarbital anesthesia. Pre-experimental and experimental conditions were identical in each group. The control animals displayed hemodilution, not hemoconcentration, due probably to trapping of large quantities of red cells within the sinusoids of the spleen. However, at the 7 and 9 hour periods when the animals had reacted from the anesthesia, and before water was given orally, their peripheral blood studies were below, rather than above, control levels. Thus we believe the observed changes during the first 24 hours after burning are due to the thermal injury alone and are not influenced by dehydration.

Actual local heat destruction of red blood cells in capillaries probably occurs at the time of contact of the burning iron with the skin. This destruction of red blood cells is thought by us to be less in dogs burned under sodium pentobarbital anesthesia, because of the trapping during actual burning of many red cells in the spleen and the resultant decrease in the number of red cells subjected to the heat. Just how this may affect the ultimate outcome in these dogs is uncertain at present. Troublesome hemolysis is present during the 4 to 12 hour samples.

Although specific data have not been accumulated, careful observations have shown that there is a greatly increased sedimentation rate in all dogs burned, beginning 1 day after burning and extending through the fourth and fifth week following the burn.

Infection was not a major problem in these dogs. It was the greatest about the tenth day after the burn. Infection began beneath the destroyed skin discs apparently being introduced through the hair follicles and slight epidermal abrasions incident to shaving the animals. Once the dead skin was elevated by the collecting pus beneath, it also separated from the normal tissues at the margins. Peeling was always aided by the dogs own cleansing maneuvers. Once the dead tissue was completely removed, the dog kept each wound clean and granulating in appearance by licking. Thus the period of maximum "uncontrolled" infection was short and not of great concern.

The primary motivation in our attempt to produce a standard burn in the dog has been our desire and need for an experimental tool with which we could study objectively the many complicating factors concerned with burns as seen in the human, especially after atomic bomb explosion. Thermal injury associated with such catastrophe is not simple; it may be accompanied by radiation injury due to gamma and neutron radiation. It is followed in some persons, depending on the position of the patient with respect to the bomb burst, by leucopenia, thrombocytopenia, infection, anemia, and malnutrition. With the standard burn preparation available, it is possible now to investigate carefully the influence of these factors, and to evaluate better the beneficial effects of certain types of therapy, in the simple and complicated burn. The value of the standard burn for such studies is already evident, and details of these researches will be published in the near future.

SUMMARY

1. A detailed discussion is presented for the technique of production of a standard burn in dogs.

2. A 20 per cent body surface burn at 60 degrees C. for 1 minute has been produced and carefully studied and analyzed in 38 dogs. In this study, weight, temperature, hemoglobin, hematocrit, red blood cell count, white blood cell count, differential counts,

total plasma protein, and blood volumes have been observed throughout the changes caused by the burn procedure.

3. No attempt at therapy in any form has been made in this study. The results of this study will be used as a "base line" against which comparisons can be made of agents and procedures which might be of value in the treatment of burns, or on the other hand which might prove detrimental to recovery processes. Complicating lesions and trauma which occur simultaneously with the burn can also be more clearly evaluated.

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ILEOSTOMY FOR ULCERATIVE COLITIS

Technique, Complications, and Management

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AT THE Massachusetts General Hospital during the past 20 years almost one-half the patients admitted with idiopathic ulcerative colitis were considered to require (Table I) surgical intervention as part of their therapeutic program. Ileostomy with or without later colonic resection was the treatment of choice (Table II). Although it has been advocated by other clinics (10, 11) partial or total colectomy at the time of ileostomy for the classical type of ulcerative colitis was not performed in this period. Although adrenocorticotropic hormone and cortisone have recently provided our therapeutic armamentarium with more potent weapons against the acute manifestations of the disease (6, 12), it is too early to tell to what degree these agents will reduce the number of patients eventually requiring surgical treatment. It is, however, doubtful if ileostomy or colectomy will be entirely abandoned until measures are found for reversing the structural changes in the colon caused by the disease.

There have been comprehensive reports published which deal with the postoperative mortality (1, 2, 8) and the later fate (9) of the ileostomy patient. Yet despite certain outstanding contributions (3, 7) more needs to be said with regard to the technique of performing ileostomy and the early postoperative management of the complications and the altered physiological processes which may result from it. It is the purpose of this report to relate an experience based upon 210 ileostomy patients seen during the 20 year period 1930 through 1949.[1]

From the Surgical Service of the Massachusetts General Hospital.

[1]The surgical responsibility for patients with ulcerative colitis at the Massachusetts General Hospital during the period under consideration was from time to time also shared with Drs. Richard H. Miller, John D. Stewart, Champ Lyons, and Francis D. Moore.

TECHNIQUE

The basic technical principles of performing ileostomy as they have been evolved by one of us (L. S. McK.) are outlined below. It is to be emphasized that of these principles the first two discussed, namely simplicity of technique and the proper location of the ileostomy, are far more important than any of the others.

Simplicity of technique. Since the primary purpose of ileostomy is to cause a remission of the disease by diverting irritating small intestinal enzymes from the inflamed colonic mucosa the simplest operative procedure has been used which will accomplish this purpose and still allow for successful management of an ileostomy bag. Field block and local infiltration procaine anesthesia supplemented by sodium pentothal intravenously has been most often used. The incisions have been as small as compatible with ready identification of the terminal ileum, and exploration of the peritoneal contents and handling of the intestines, particularly the diseased colon, have been avoided.

Desired location. For proper function of an ileostomy it is important to select a point in the right lower quadrant of the abdomen as far as possible from irregularities of surface such as the anterior superior iliac spine, the umbilicus and the symphysis pubis, which may cause leakage of discharge between the ileostomy bag and the skin. Since abdominal wall topography varies in different individuals the skin area available under the aforementioned considerations may occasionally be so small that a point equidistant from the landmarks mentioned may bring the bag too close to the anterior superior iliac spine or to the symphysis pubis. This has been avoided and the stoma has been placed under such circumstances closer to the umbilicus than to the bony prominences.

TABLE I.—ULCERATIVE COLITIS—M.G.H.—1930–1949—TOTAL PATIENTS

	No.	Incidence—%
Treated by definitive surgery	240	48.4
Not treated by definitive surgery	255	51.6
Total	495	

Less important but useful points in technique are the direction of the incision, the exposure, the identification of the terminal ileum, the creation of the stoma and its postoperative management.

Incision. A longitudinal rectus muscle splitting or a transverse incision have both been used. The latter has several minor points of advantage, namely that it does not transgress the lines of skin elasticity, if it becomes infected lateral drainage is more convenient, and if there is any keloid or other irregularity of the abdominal wall caused by the operative scar which will make less secure the union between the ileostomy bag and the skin, this weak area is, because of the tendency of the discharge to flow downward, less vulnerable laterally than inferiorly (Fig. 1).

Exposure. The terminal ileum is identified by inserting two abdominal wall retractors and moving the incision about until the ileocecal region is seen. Frequently preliminary study of the gas shadows in an abdominal x-ray film has furnished a useful preoperative idea of the location of the cecum. Occasionally it has been necessary to use a smooth instrument to move the intestinal loops aside, but pinching of the serosa or handling of it with gauze has been avoided.

TABLE II.—ULCERATIVE COLITIS—M.G.H.—1930–1949—DEFINITIVE OPERATIONS IN 240 PATIENTS*

Operations	No.	Deaths	Mortality—%
Ileostomy	210	37	17.6
Subtotal colectomy	113	10	8.8
Proctosigmoidectomy	49	2	4.0
Total colectomy	7	0	0
Ileocolostomy without resection†	10	1	10.0
Primary colectomy†	8	1	12.5
Colostomy**	9	6	66.6
Exploratory laparotomy**	3	1	33.3
Totals	240	58	24.1

*A few patients had part of their surgery elsewhere.
†All patients with localized disease.
**Very ill patients in whom diagnosis was not clear.

Identification of terminal ileum. Positive identification of the terminal ileum has been accomplished without necessity of handling the intestines by searching for the ileocecal fold (the bloodless fold of Treves). It is a crescentic peritoneal fold, containing between its layers varying amounts of adipose tissue and running longitudinally for 4 to 8 centimeters on the antimesenteric border of the terminal ileum onto the cecum (Fig. 2). No other loop of small intestine presents a similar structure on its antimesenteric aspect. It is furthermore helpful to remember that the terminal ileum is the loop of small intestine which habitually lies most inferiorly in the peritoneal cavity and so approaches its termination in the cecum by running upward from the pelvis.

The creation of the stoma. For purposes of convenience in later management the ileostomy stoma is best arranged as a simple single rosette of terminal ileum which emits the ileal discharge and is referred to as the "end" ileostomy. Such a stoma is created by dividing the ileum and performing a suture turn-in of the distal cut end of the ileum ("ileal stump"). Fear that this procedure might be dangerous because of failure of sutures to hold in the fragile bowel, or of a later "blow out" from the back pressure of ileocolonic secretions has been unfounded. Of our 210 cases 137, or 65.2 per cent, had end ileostomies of this type. In only one did the ileal stump cause symptoms, and in him they were low grade and were eventually cured by resection of the dilated stump.

In our early experience many loop ileostomies were done. Of recent years, however, although loop ileostomy is still used, and we feel indicated, in desperately ill patients, the end ileostomy is applied to the majority. The afferent limb is brought out through the incision which is closed in layers with interrupted sutures leaving a palpable opening for transmission of the bowel which will no more than easily admit the finger. No sutures are taken in the bowel to unite it to the abdominal wall lest damage be done which will cause a fistula at that point. About 3 centimeters of ileum is brought beyond the skin, a glass tube or catheter is placed in the stoma to lead the dis-

charge away (Fig. 3)[1], the stoma is encircled with dry gauze to which it rapidly becomes adherent and, being thus incorporated as part of the dressing, is prevented from prolapsing to the outside.

Postoperative management. The tube is allowed to drain by gravity into the bedside bottle for a period of 5 days. At this time an adherent ileostomy bag is applied directly over the skin sutures. The patient is thus spared a period of demoralizing, uncomfortable, and malodorous contamination of the skin, the apparel, and the bed, with intestinal contents. Since the stoma initially is, and during the first 2 postoperative weeks becomes increasingly more bulky than is true later in the postoperative course, the bag which is applied early must have an opening larger than is eventually most practical. This can be circumvented by using either a temporary inexpensive bag such as is recommended by Haase (Fig. 4), or by a permanent bag with a large opening which is later made smaller by attaching a rubber washer with a smaller opening as has been recommended by Dixon (Fig. 5).

POSTOPERATIVE COMPLICATIONS

A discussion of the postoperative complications, including the postoperative mortality, is based upon our experience with 210 ileostomy patients, 37 of whom (or 17.6 per cent) died in the postoperative period (Table III). One hundred ninety-seven of the ileostomy operations were performed at the Massachusetts General Hospital and 13 were performed elsewhere. The postoperative mortality and the incidence of nonfatal postoperative complications have been in direct relation to the degree of illness of the patient at the time ileostomy was done. Thus Table IV shows that 23 of the 37 deaths were in patients in whom medical therapy had been unable to effect improvement prior to ileostomy. This most important of all considerations, namely the selection of the proper time for ileostomy and the technique of preoperative preparation, are not within the scope of this paper and will not be further considered.

[1]Devised by Dr. Claude McGahey.

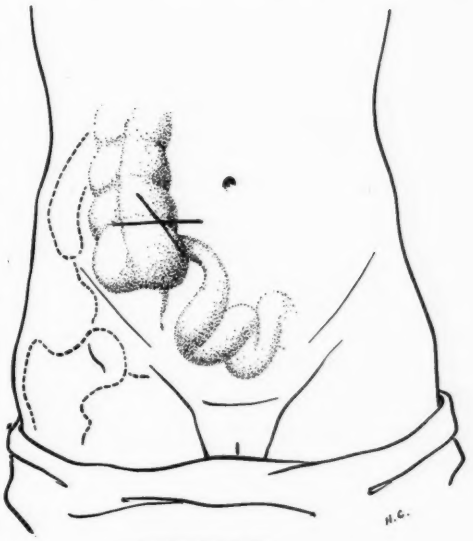

Fig. 1. Incisions for ileostomy.

Causes of mortality. Of the 37 patients who died following ileostomy (Table V) the 17, or 45.9 per cent, who died of peritonitis, toxicity, and colonic hemorrhage must be considered as succumbing to progression of their disease which was not checked by ileostomy. The 14, or 37.8 per cent, who died of ileostomy dysfunction, pulmonary embolism, and pneumonia did so from complications attributable to the operation. Many of the deaths in both

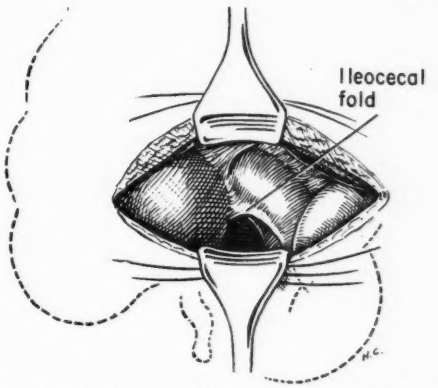

Fig. 2. Identification of the terminal ileum. The ileocecal fold is demonstrated.

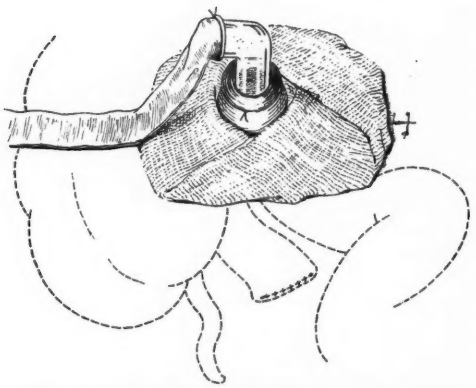

Fig. 3. A useful type of apparatus for the immediate postoperative period. The glass tube terminates in that portion of ileum which is external to the abdominal wall.

these groups we now believe could have been prevented. In support of this the total mortality from ileostomy fell from 26.3 per cent in the first half of the study to 12.6 per cent in the latter half.

Failure of remission of the colitis. In 140, or 66.6 per cent, of the patients the disease underwent a complete remission following ileostomy. Twenty, or 9.5 per cent, died of complications of the ileostomy or of intercurrent diseases. In the remaining 50 patients, or 23.9 per cent, ileostomy did not accomplish a complete remission. Seventeen of them went on to die of their disease and the remaining 33 were submitted to "urgent" colectomy, 24 of whom were saved by the procedure. Of the 9 patients who were not saved by urgent colectomy, 6 died of peritonitis and 3 of "toxicity." Our opinion is that earlier decision for ileostomy in the patients showing signs of peritoneal irritation would have prevented a few, but not all, of the above postileostomy deaths and that earlier application of urgent colectomy would have prevented 4 of the 9 deaths which followed that procedure.

Hemorrhage from the colon. Bleeding by rectum of a significant degree is not an infrequent occurrence following ileostomy. It occurred in 35, or 16.0 per cent, of the patients and was of an alarming degree in 6. It is most likely to occur in those who have had considerable blood in the stools prior to ileostomy. The cause is not always clear. It is only occasionally associated with a significant hypoprothrombinemia, is perhaps a phenomenon akin to hemorrhage from the chronically distended bladder which is suddenly decompressed. It

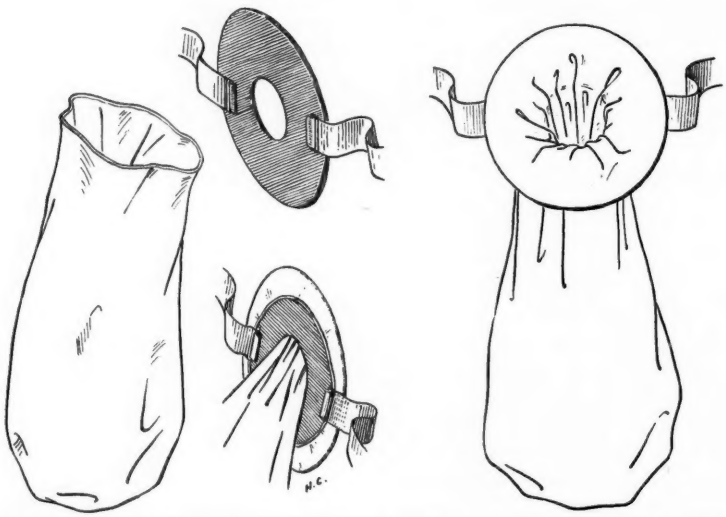

Fig. 4. A simple bag for the early postoperative management of an ileostomy before the permanent bag is procured. Any other temporary device which is inexpensive and will adhere to and so protect the skin is satisfactory.

will usually subside spontaneously in a few days. It was necessary to perform an urgent colectomy in order to control bleeding in one patient, however, and another in the early years of the series died from hemorrhage before we had learned the importance of aggressive supportive therapy for this complication.

Perforation of the colon. Perforation of the colon was found in 5 patients with ileostomy coming to autopsy. In 3 of them the perforation had occurred before ileostomy. In the 2 others it arose after ileostomy, apparently due to unbridled progression of the disease rather than to trauma inflicted at the time of operation.

Peritonitis without gross perforation. There were 3 additional patients who died of peritonitis following ileostomy in whom no intestinal perforation could be found at autopsy. It was presumably due to spreading of organisms through microscopic openings in the colonic wall.

Perforation of the ileum. There were 3 patients in whom perforation of the ileum occurred following ileostomy. All 3 were patients who were acutely ill, the disease being uncontrolled by medical measures prior to ileostomy. In 2 of them some degree of ileostomy dysfunction existed. In both it was recognized late. In the third patient, desperately ill with colitis, a localized perforation occurred a few days after ileostomy in what at autopsy appeared to be normal ileum 40 centimeters proximal to the stoma.

It is our impression that the two most important factors which will avert ileal perforation after ileostomy are the selection of the proper time for the operation in the course of vigorous medical therapy and the early recognition and agressive management of ileostomy dysfunction.

Ileostomy dysfunction. Early fixation of the ileum by the abdominal wall and later contraction of the stoma are the most harmful features resulting from ileostomy. Although complete obstruction rarely occurs at the ileostomy stoma, partial obstruction is common. It was seen in 130, or 62 per cent, of our patients. It is characterized by cramp-like

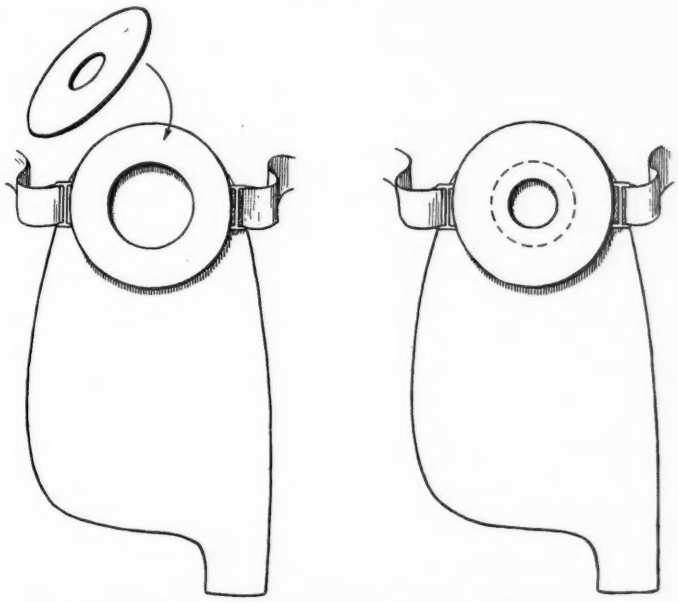

Fig. 5. Device for decreasing the size of the opening in a permanent ileostomy bag necessitated by shrinking of the stoma with the passage of time.

TABLE III.—ULCERATIVE COLITIS—M.G.H.—1930–1949—IMPORTANT POSTOPERATIVE COMPLICATIONS FOLLOWING ILEOSTOMY IN 210 PATIENTS

	No.	Per cent
Mortality	37	17.6
Failure of remission of colitis	49	23.3
Hemorrhage from colon	35	16.6
Peritonitis	11	5.2
Perforation of colon 5		
Perforation of ileum 3		
Without perforation 3		
Ileostomy dysfunction	130	61.9
Ileostomy fistula	15	7.1
Ileostomy prolapse	30	14.3
Chronic 28		
Acute 2		
Wound infection*	27	12.9
Contact dermatitis*	13	6.1
Hernia*	4	1.9

*Only the more severe instances recorded and no complete follow-up.

pain and, paradoxically, an increase in the volume of ileostomy discharge. The discharge becomes watery. In the severe cases vomiting occurs and ileostomy diarrhea becomes so severe that serious water and electrolyte deficiencies may result. The first systemic manifestation may be sudden evidence of profound fluid and electrolyte imbalance as manifested by a shock-like state. In the later stages abdominal cramps may cease and thus further tend to cloud the diagnosis. Figure 6 presents the basic data on one patient who required intravenous fluid replacement of up to 7 liters a day in order to repair the losses brought about by this diarrhea.

The patient may appear listless, toxic, and under the influence of an exacerbation of the inflammatory process in the colon or of a spread of it to the ileum. The former can be ruled out by the absence of copious rectal discharges of bloody and purulent exudate which

TABLE IV.—ULCERATIVE COLITIS—M.G.H.—1930–1949—INDICATIONS FOR ILEOSTOMY IN 210 PATIENTS

Indications	No.	Incidence—%	Deaths	Mortality—%
Emergency: disease out of control before ileostomy	65	30.9	23	35.3
Urgent: disease partially controlled before ileostomy	92	43.8	13	14.1
Elective: intractable chronic disease or premalignant lesion	49	22.3	1	2.0
Record not clear	4	1.9	0	0

usually accompany such an event. Although involvement of the terminal ileum in the disease process was noted in 31, or 20.4 per cent, of 152 ileocolonic specimens from patients with ulcerative colitis examined in the department of pathology, spread of the disease to the ileum proximal to the ileostomy has, in our experience, rarely, if ever, occurred. It is true that if ileostomy dysfunction is allowed to continue, ulceration of the ileum near the ileostomy may ensue, as was demonstrated in 11 of our patients, and be evidenced by bleeding from or perforation of the ileum. But in the 8 patients of the 11 with structural changes in this part of the ileum who did not succumb, the inflammatory process subsided following spontaneous or operative relief of obstruction to the ileostomy. We have, therefore, come to believe that such ulcerating processes are secondary to the dilatation and hyperactivity of the obstructed bowel rather than primarily the same disease as ulcerative colitis.

The importance of making the diagnosis of ileostomy dysfunction is shown by the fact that in 7 of the fatal cases the patients suffered from ileostomy dysfunction as one of the major causes of death. Furthermore, in the 3 deaths from perforation of the ileum it could not be entirely excluded as one of the factors bringing about this serious complication. We believe that the single, most important safeguard against overlooking the diagnosis of ileostomy dysfunction is to record the daily amount of ileostomy drainage. Any amount over 1,000 cubic centimeters a day is suspicious of trouble and should lead one to more careful measurements of all elements of the fluid balance.

Although it may first occur on the day following operation or not until after the third week, ileostomy dysfunction is more frequently noticed at the middle or end of the second week. The cases which occur early are usually due to interference with the normal flexibility and peristaltic action of the ileum by the comparatively rigid abdominal wall, those which occur late, the result of a constricting band of scar tissue which replaces the granulation tissue on the serosa of the exposed ileum and draws the mucosal margin down toward the skin. Frequently a com-

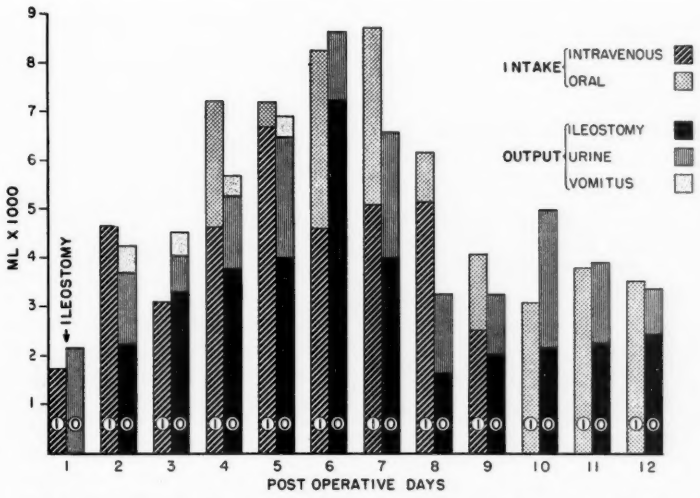

Fig. 6. Case 591431. Diagram of fluid intake and output in the 11 days after ileostomy. The large output via ileostomy of up to 7,000 milliliters in 24 hours and the vigorous measures necessary to repair it are demonstrated.

bination of the two mechanisms is at play. Frequently also, however, the local factors determining which patient will suffer most from this difficulty seem hard to define. Many patients have no trouble when the stoma is so small that it will not admit the finger and others with large openings have severe symptoms. There is undoubtedly a great variation in the irritability and tolerance of the intestines of different individuals to similar degrees of obstruction.

In most instances the ileum becomes adapted to the abnormal situation over a period of weeks, and symptoms tend to subside spontaneously. We feel that the tendency for ileal discharge to become thicker as months go by is more the result of the progressive adaptation of the ileum to this partial obstruction than to some mysterious instinct on its part to take over the function of a resected colon, an explanation of this phenomenon which has been invoked by some.

Measures for managing ileostomy dysfunction are varied and must be adapted to the individual case. Technical modifications have been devised to forestall it, chief among which is the advocation by Dennis of the excision of a circular wafer of skin of the abdominal wall to avoid constriction at this point as occurs when the conventional incision tends to heal. This has not been uniformly successful in our hands.

The "early" cases characterized by lack of pliability of the ileum generally respond to catheterization and conservative methods. The later ones usually require division or excision of the constricting band of granulation or scar tissue.

If the patient complains merely of cramps and has no distention, vomiting, or ileostomy diarrhea, the institution of gentle digital di-

TABLE V.—ULCERATIVE COLITIS—M.G.H.—1930–1949—PRINCIPLE CAUSE OF DEATH FOLLOWING ILEOSTOMY IN 37 CASES (27 AUTOPSIES)

	No.
Peritonitis	11
Toxicity	5
Colonic hemorrhage	1
Ileostomy dysfunction	7
Pulmonary embolism	3
Pneumonia	4
Disseminated tuberculosis	1
Amyloidosis	1
Undetermined	4
Total	37

Fig. 7. Plastic operation to ileostomy for early postoperative dysfunction. The release of the contracting collar of scar tissue which forms where the granulations of the serosa and the edge of the everted mucosa join is the most important objective of this maneuver.

latation of the stoma (never enough to cause serosal bleeding) and a low residue diet are indicated. If distention and vomiting occur, a soft (preferably old) No. 26 French rubber catheter should be inserted into the stoma and an estimate made, from the intestinal contents procured from the ileal loop just within the abdominal wall, as to whether that loop is dilated. In such patients as much as 1,000 cubic centimeters of fluid is frequently immediately obtained by this means and the diagnosis thus confirmed. The catheter is allowed to remain in place for 4 hours, removed for 1 hour, and then reinserted for another 4 hour period. This intermittent schedule is used in order to prevent erosion of the bowel wall by the tip of a catheter which may exert pressure against one spot for too long if it is allowed to remain continuously in place.

If the ileostomy does not tend to work spontaneously after 2 or 3 days of such a regimen a plastic operation on the stoma should be done. We have found this necessary 95 times in 75, or 35.7 per cent, of patients receiving ileostomy, or in 57.7 per cent of those developing dysfunction. In the majority of the patients it was accomplished by dividing radially in two or three areas the contracting collar of granulation tissue which forms over the extracorporal aspect of the stoma (Fig. 7). It can be done without anesthesia with the patient in bed, having in mind to avoid that area on the circumference of the stoma where the ileal mesentery resides. Only one or two blood vessels ordinarily need ligature during the procedure. Occasionally a more formal operative procedure is necessary, in the form of a resection of the tight ring of skin around the base of the stoma, under anesthesia in the operating room (Fig. 8). In a few patients complete pliability of the stoma was not secured by either of these methods, and a dissection and "freeing-up" of the stoma through all layers down to the peritoneal cavity was necessary (Fig. 9). Such a procedure fortunately does not simply re-create the original situation and a similar danger of later obstruction. The propulsive powers of the terminal ileum, despite having been demonstrated inadequate to the existing degree of impedance have by it nevertheless been stimulated to a point that is sufficient to avoid trouble after the secondary procedure.

Occasionally a combination of the above methods or the repeated use of one or more of them will be necessary before the intractable cases reach a state of equilibrium. The important thing, however, is that such an equilibrium was invariably attained in those cases recognized early.

Ileostomy fistula. Fifteen, or 7.1 per cent, of the patients upon whom we performed ileostomy developed fistula of the ileostomy stoma (Fig. 10). Such fistulas have been located at some point between a level just deep to the skin and one-half way between the skin and the stomal opening. Through them varying proportions of the total ileal stream are at first discharged. They tend to increase in size so that eventually all of the stream passes through the tract. When the fistula occurs deep to the surface of the skin an infection of the subcutaneous tissues develops. If it occurs superficial to it no untoward effects are experienced other than that the collection of the discharge and the protec-

tion of the skin against it become more difficult.

Fistulas are caused by damage to the bowel which may occur at the time of ileostomy from traumatic handling, from injury with an instrument or a needle at the time of suturing the abdominal wound, or may be incurred later from the catheter, the rim of an ileostomy bag, or a too vigorous digital dilatation of the stoma. Since adherent bags with openings tailored to the size of the ileostomy have been used, late fistulas just superficial to the skin level resulting from damage by the rim of the bag have occurred more frequently. If the bag opening is not exactly centered, or if the adhesive becomes loose and permits a shearing action of the bag rim against the stoma there is danger.

Preventive measures against fistulas are gentle handling of bowel, careful suturing of the abdominal wall, and the avoidance of sutures uniting ileostomy to abdominal wall. Bag openings should be larger than advocated by the makers. We allow a full 5 millimeter clearance between the stoma and the margin of the opening at all aspects of the circumference of the stoma. The dermatitis arising in the small ring of skin so exposed is usually of no consequence.

Once a fistula has occurred an operative procedure is indicated. Direct suture of the fistula has been unsuccessful in all cases in which it has been tried. If it is below the skin level the resulting wound sepsis should be surgically drained and a major ileostomy revision done, the stoma being amputated distal to the fistula and the bowel being mobilized through all layers of the abdominal wall and pulled out so that more proximal normal ileum is utilized for the ileostomy (Fig. 9). If the fistula is distal to the skin a simple incision through all layers of the stoma uniting the fistula and the lips of the normal opening should be made. This creates a single opening through which the discharge emerges. The resulting stoma is deformed in that it has a longer wall on one aspect than another. Experience with the individual case only will tell whether this deformity will interfere with proper management. Frequently the only additional maneuver necessary is the trimming away of part of the redundant bowel wall. In a few cases in which the fistula is close to the skin it engenders leakage between bag and skin. If this occurs then a major ileostomy revision as outlined for deeper fistulas must be done at an elective time.

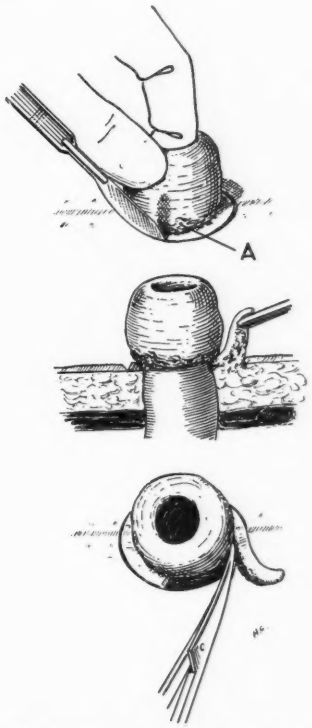

Fig. 8. Plastic operation to ileostomy for dysfunction occurring after the second postoperative week. The release by excision of the constricting scar which formed as a result of the mucosal edge being pulled down to the skin is the objective of this procedure.

Ileostomy prolapse. Mild chronic prolapse of the ileum with eversion of the lumen out through the ileostomy stoma is a common occurrence. In many patients it is asymptomatic and, therefore, not recorded. The prolapse reached proportions which demanded relief of symptoms in 28, or 13.3 per cent, of our cases. Differences in the size of the abdominal wall defects were not apparent when this group was compared with those not having prolapse. It is our opinion that

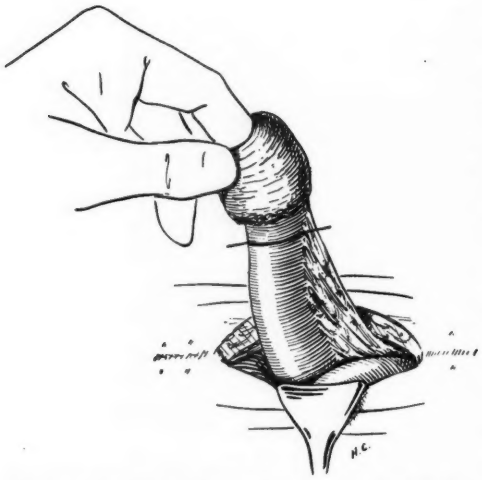

Fig. 9. Plastic operation to ileostomy for dysfunction which, due to excessive fibroplasia of the wound, persists after the simpler procedures have been tried. The stoma is amputated and a new ileostomy created in the same area.

there is some inherent peculiarity of the ileum itself or of its mesentery which renders certain patients more prone to develop it. This opinion is based on the fact that in all 5 patients who underwent local ileostomy revisions in which the size of the abdominal ring was decreased there were recurrences taking place within a few months after operation. Suspension of the ileum from within by suture fixation of the serosal surface or of its mesentery to fixed areas of parietal peritoneum has been similarly disappointing.

Prolapse is most often first noticed between 4 and 6 months after discharge from the hospital. The onset is usually gradual and the condition slowly progressive as the mesoileum becomes stretched.

In a few of our patients, the initial appearance of prolapse was more dramatic and characterized by the sudden protrusion of a foot or two of ileum, which, in 3 cases, became incarcerated. Incarceration is due to the fact that early reduction is not performed. If such an acute prolapse is not reduced promptly it becomes edematous from constriction of the venous return at the abdominal ring and traction on the mesenteric veins. Reduction then becomes doubly difficult.

We believe that in the majority of cases the treatment of chronic ileostomy prolapse should be nonsurgical. If the prolapse is small and not progressive nothing need be done. If it is larger an improvised belt or girdle worn around the body outside the bag and, by means of a pad, exerting localized pressure against the stoma through the bag wall usually provides a sufficient restraint. If, despite conservative measures the patient continues disabled and wishes the situation corrected, the ileostomy may be transplanted to the upper abdomen. This was curative in the one case of our series so treated.[1] It is technically simple to perform due to the fact that the ileal mesentery has been extensively stretched by the prolapse.

The management of incarcerated prolapse must be more radical. If the bowel is not badly discolored and has not ulcerated or bled, a period in the supine head-down position under sedation and followed by gentle taxis on the prolapse is indicated. If the bowel is obviously not viable it must be resected and a new ileostomy created forthwith. We have seen 2 patients in whom the prolapse has been incarcerated in an adherent bag by the constricting rigid ring of the bag opening. In 1 of these patients we were able to procure a hacksaw and remove the ring before the bowel became nonviable. In the other we were not and resection was necessary as an emergency procedure. This is an additional reason for recommending that the bag opening be made 1 centimeter greater in diameter than the diameter of the stoma.

Another type of prolapse was found twice in this series. It consisted of the prolapse or evisceration of the ileostomy and a foot or two of the ileum without eversion of the lumen out through the abdominal wound in the early hours after ileostomy before it had become adherent to the abdominal parietes. It is a complication which those who advocate suturing of the ileum to the abdominal defect are undoubtedly able to prevent by that maneuver. In both of our patients, however, the ileum was replaced surgically immediately with no ill effects.

[1]Operated upon by Dr. Francis D. Moore.

Paraileostomy hernia. In 2 patients the ileal loop, which had begun to prolapse in the manner described, did not protrude beyond the skin but passed into the subcutaneous layers of the abdominal wall. This was recognized in both patients as being a certain source of a later hernia, but since ileostomy function was not interfered with at that time, nothing was done. One of these patients has subsequently come to repair of the hernia and transplantation of the ileostomy to the left lower quadrant; the other has a paraileostomy hernia but does not wish operative treatment. There are 2 other patients who have presented themselves with paraileostomy hernias in whom the occurrence of acute hernia was not recorded in the early postoperative period. It is probable, however, that it took place at that time but was unrecognized. For one of them likewise ileostomy transplantation and primary herniorrhaphy were done with success whereas the other has decided against surgical treatment. It is of interest that in these 4 patients with hernia, obesity has been a problem as it has been in several other ileostomy patients without hernia and is good testimony that an ileostomy need not interfere with nutrition.

Wound infection and wound separation. In many cases minor and insignificant degrees of wound infection take place in the few millimeters of the incision nearest the ileostomy. Major wound complications occurred in 25 of the patients. These were divided as follows: wound infection alone 6, wound separation alone 12, wound infection and separation 7. One of the causes of wound sepsis has been mentioned, namely ileostomy fistula, and was responsible for 6 of the cases. Certain instances of wound separation seem to be more associated with failure of healing than with infection introduced at the time of operation. In such cases the dermal and subcutaneous layers have suddenly separated down to the fascia. This occurred in 12 patients at intervals of from 8 days to 4 months after ileostomy. It is more likely to occur in patients who have ileostomy at a time when the ulcerative colitis is out of control than in the less sick group, and of these it is more often found in those in whom ileostomy does not induce a complete remission of the colonic disease.

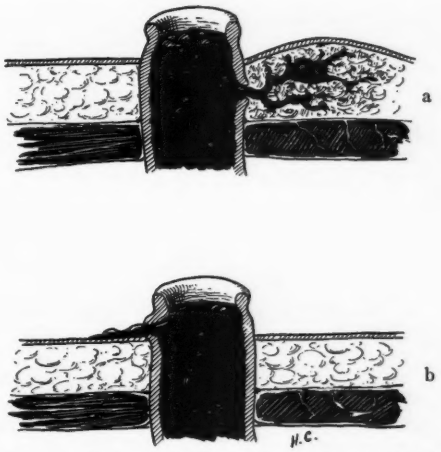

Fig. 10. Ileostomy fistula. A fistula deep to the skin surface causing a wound abscess, a, and one superficial to it, b, are depicted.

Following such separation resuture cannot be done. The ileostomy discharge is best led away from the skin by a catheter and vigorous repair undertaken of systemic nutritional deficiencies which may be more evident clinically than by laboratory test. In most instances such measures will cause the area to form healthy granulations and heal by second intention. Occasionally an anaerobic type of infection will occur and the wound will increase in size. This occurred in one of our patients who went on to die despite cautery excision of the area.

Dermatitis. Some degree of skin irritation resulting from contact with the ileal discharge results in all patients with ileostomy. Since the advent of the adherent bags and our appreciation of the importance of their application as soon as the discharge is no longer controlled by a catheter or stomal tube, the incidence of severe irritation has become much less. Only 11 patients are recorded as presenting major problems in dermatitis. The only permissible modern excuse for its occurrence is that the patient's bag has become lost or damaged or that because of severe irregularities in the abdom-

inal wall it cannot be worn. Then the use of an intermittent catheter, or a nonadherent bag placed on a cotton pad covering the irregular skin will avoid this complication.

The treatment of extensive dermatitis once it has occurred is difficult. Our best success has been with the immediate procurement and application of an adherent bag directly over the inflamed skin. The application may be uncomfortable but unless the surface is so moist that the adhesive agent will not stick, the underlying dermatitis will quickly disappear due to the removal of the irritating agent from the skin. If such a bag cannot be worn the best course is to place the patient prone in bed on an elevated mattress or Bradford frame provided with a hole through which the ileostomy drainage can drip by gravity without contacting the skin. This seemingly drastic treatment is effective in a few days and pays dividends provided a proper bag is available for application when it is discontinued.

If neither of these methods is possible a protective agent for the irritated abdominal skin must be used. The use of such agents is customarily given first place in priority of therapy for such dermatitis. We are intentionally giving it low priority because we earnestly feel that a better and a more rapid result will be attained by the first-mentioned methods. An ideal topical application which will protect the skin of every patient against ileal discharge has not yet been brought forward. We have, however, used with best satisfaction rubber waterproof cement over a wide area of skin. Other less effective applications are petrolatum, aluminum paste, Fuller's earth, kaolin, yeast powder, and aluminum hydroxide gel. Before the advent of adherent bags each patient had his own particular preparation which he was convinced worked best for him. There is no physical handicap in medicine in which the element of non-analyzable individual preference is more evident than in the management of an ileostomy stoma by its owner.

Surgical correction of irregularities of the skin surrounding the ileostomy is essential in instances in which they interfere with the adherent properties of the bag and may be necessary after final healing of an infected or separated wound.

Discussion. The experience related demonstrates that a recognition and understanding of the complications which follow ileostomy are essential to the safety of the operation. The total surgical mortality for all operations in ulcerative colitis at our hospital has been 23.3 per cent. One hundred sixty-one of the 210 patients having ileostomy survived their surgical program. The mortality following ileostomy in the second of the decades studied was 12.6 per cent as against 26.3 per cent for the first decade. This decrease in mortality has resulted, we believe, to some extent from newer techniques of anesthesia, transfusion, and antibiotic therapy but in much larger measure from a proper timing of surgery and a wider experience in the details of the early postoperative management of the ileostomy. This theory is demonstrated by the fact that, of the 65 "emergency" ileostomies performed, 34, or 52.3 per cent, were done in the first decade, which period contains only 34.7 per cent of the total ileostomies.

A previous study of the social and economic status of 104 of these patients (9) revealed that 97 per cent were working as housewives, in schools, or were gainfully employed and that only 33 per cent felt that the ileostomy interfered in any way with their social lives. Eighty-eight per cent of the patients were in good health. There were no suicides.

The most important consideration from the patient's point of view is that constant vigilance and attention to detail will in the end mean *total rehabilitation* to a state where the individual is independent of medical supervision. Neglect of such details spells, if not a mortality, at least partial invalidism, a state in which the patient is unable to sever the bond connecting him to the hospital or to his medical adviser.

SUMMARY

1. Experience with the technique and the early postoperative management of 210 ileostomies for ulcerative colitis is presented.

2. Simplicity of technique and the proper location of the incision, together with less important technical details, are emphasized.

3. The postoperative complications of mortality, failure of remission of the colitis, perforation of the colon, ileostomy dysfunction, perforation of the ileum, ileostomy fistula, ileostomy prolapse, wound infection and separation, dermatitis, and postoperative hernia are considered. Suggestions as to management of these complications are made.

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THE TREATMENT OF EDEMA OF THE ARM

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THE management of swelling of the arm following axillary surgery and axillary vein thrombosis has been given scant attention in the medical literature. Several plastic surgical procedures have been devised with rather indifferent success (2, 5). Characteristically, this condition is progressive, disabling, and often painful. Some patients first notice swelling within 6 weeks of operation, but others have no appreciable edema for as long as 5 or more years. In some, the upper arm swells at first, followed in turn by swelling of the forearm, wrist, hand, and fingers. In other patients, swelling of the hand is noticed first with progressive enlargement of the arm. The edema may increase with great rapidity or develop through the years and, in a considerable number of patients, it eventually leads to massive elephantiasis with spontaneous development of blebs in the skin which may rupture and drain (Fig. 1, NYH No. 411291). Neuralgia which is persistent and disabling may be a major complication. It is frequently related to the increased weight of the arm and often disappears when the swelling is reduced by treatment. The psychological effect of a persistently swollen hand and arm is very great. Many women become seriously depressed. To a considerable degree, this reaction has been due to the discouraging attitude of physicians in general toward this problem. One patient (Case 1) became a recluse for several years because of her appearance. Reduction of the swelling under treatment has enabled her to abandon her seclusion.

There has been much speculation as to the etiology of the edema. Halstead believed that the swelling occurred only in cases complicated by postoperative infection. In cases in which no infection was demonstrable, he stated that "the infection may be so mild in degree as to escape observation." Holman and coauthors have recorded experience confirming Halstead's position. Forty-six of a total of 59 of their patients with infection developed swollen arms, while only 7 of the 41 patients without recognized infection were affected in this manner.

A somewhat different theory is offered by Veal who believes that many cases of postmastectomy swelling are associated with axillary vein thrombosis and supports his thesis by x-ray plebograms. He has demonstrated that the axillary vein is frequently displaced and angulated by the operation. In the position of rest with the hand at the side, the lumen may be greatly narrowed in its new location.

Our postmastectomy cases have not shown clinical signs of axillary vein thrombosis, namely dependent cyanosis, increased venous pressure, and development of collateral veins. In one patient with massive elephantiasis (Fig. 1), we performed a phlebogram which showed a normal axillary vein. However, the diodrast injection produced an acute phlebitis with an axillary venous thrombosis followed by exacerbation of her symptoms.

Cases of recurrence of malignant disease with blockage of lymph channels and lymph nodes have been excluded from our study. Such cases have a very bad prognosis, and do not respond to medical management. In Holman's series of 100 cases, recurrence took place in only 5, 3 with slight or moderate swelling and 2 with no swelling. Recurrence of disease must be considered in each case, and careful examination made including roentgenogram of the chest. The axilla and supraclavicular areas of both sides and the opposite breast should be palpated expertly. At times recurrent disease will not be detectable for several months after swelling of the arm commences. One such patient responded at first to medical management, but 6 weeks later swelling recurred. Metastatic areas appeared in the film of her chest, and she died shortly thereafter.

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Theoretically, the fact that surgical extirpation of lymph channels and nodes, incident to radical mastectomy, reduces the number of lymph vessels draining the arm would seem to be a sufficient explanation for the development of edema. Such an explanation, however, is not supported by the animal experiments of Reichert. Working with dogs, Reichert divided all tissues of the hind limbs except the bone, femoral artery, femoral vein, and nerve trunks. He then sutured the tissues together. Edema of the leg did not occur. By the fourth day, new lymphatics could be shown to have crossed the line of suture. By the eighth day, "regeneration" was "physiologically adequate." It might be noted, however, that both Guthrie and Gagnon and Devenish and Jessop have expressed the opinion that the experiments above cited and radical mastectomy are not comparable procedures. They believe that the extirpation of the whole mass of regional lymph nodes and varying lengths of the main lymphatics of the arm cannot be compared to a clean circular cut across a limb.

The development of scar tissue with subsequent contraction must also be considered as an important factor in some patients. Patients with long standing massive edema develop a firm jelly-like subcutaneous tissue which does not drain off even on maximal elevation. Such a patient, already referred to, is shown in Figure 1. While the superficial soft edema does drain on elevation, only slight improvement results. Such patients sometimes develop blebs in the arm from which serum exudes.

Without reaching any set conclusion with respect to the conflicting theories here discussed, we have attempted to treat swollen arms by the judicious application of sound physiological principles. Gravity, compression, massage, and in some cases dehydration have been used.

GRAVITY

The edema of the arms is almost invariably reduced by prolonged elevation. Although the major lymphatic channels and nodes have been surgically removed or damaged by x-ray, drainage through collateral lymphatic channels can occur with elevation of the limb. The decrease in venous pressure which occurs when the arm is elevated may also be a major factor. Keeping an arm in a position of marked elevation for many hours is very tiring for the patient unless a frame with

Fig. 1. Postmastectomy swelling. Massive edema of right arm, slowly progressive for a period of 15 years after mastectomy.

Fig. 2. Case 4. Postmastectomy swelling. Arm is suspended in elevation with counterweights. The tension line is attached to the finger of a snug-fitting glove.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 3. Case 1. Postmastectomy swelling. Edema of hand, fingers, and arm at beginning of treatment.
Fig. 4. Case 1. Elastic sleeve in place.

Fig. 5. Case 1. Three months later. Edema has cleared from fingers and hand. Some fullness remains in the forearm.

counterweights is employed. An ordinary fracture frame (Balkan) may be used. A line is run from the hand up over a pulley, then horizontal for several feet, and over a second pulley to a counterweight. Similar results can be achieved by screwing two pulleys in the ceiling over the bed. By the use of such an arrangement, the patient may rest in comfort with the arm at maximum elevation. This frame even permits movement in sleep, and no muscular effort is required to hold the arm up. For patients who travel and need a portable arrangement, the apparatus shown in Figure 2 which adjusts to the bedpost may be used. It is very convenient, simple to operate, and economical.

The method of attaching the patient's hand to the rope is an important problem. If a circular bandage or wrapping of any sort is used it produces, under tension, a tourniquet effect and constricts the return flow from the tissues. A more satisfactory method consists of sewing the rope to the tip of the middle finger of a well-fitting glove. In this way, the tension is distributed throughout the hand, and circular constriction is avoided.

We have seen one patient who had arterial insufficiency in the hand in addition to postmastectomy swelling (NYH No. 474868). The palmar arch was occluded as a result of arteriosclerosis obliterans and the added traumatic factor of bearing weight on a cane. With the arm in dependency, the tissues of the fingers received barely enough blood flow to support their life. Trophic changes were present. Nail growth was slow. With the hand elevated, the fingers became a cadaverous white. In this case, elevation could not be prescribed as it might have produced gangrene. This case emphasizes the need for examining the arterial supply of the arm before elevation is prescribed. If the radial pulse is of good quality, the palmar arches and the ulnar arteries are intact, as demonstrated by the Allen test, and if there is no pallor on elevation, one may assume that the arterial supply is adequate. Oscillometric readings will give additional information. One must also be sure that the arterial flow is adequate in the position of elevation. The pulse quality may be impaired in various positions of the arm (Wright, 9). The arterial supply should therefore be checked to be sure that it is not diminished with the patient reclining and the arm elevated to the position to be prescribed for the treatment of the edema.

COMPRESSION

Edema fluid drains out of the arm when the arm is suspended in elevation. When the

Fig. 6. Case 5. Postmastectomy swelling. Onset of reatment. Note swelling of hand and arm.

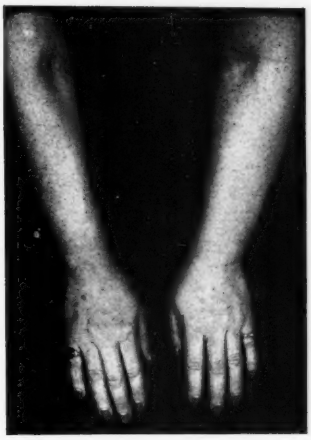

Fig. 7. Case 5. Two months after onset of treatment. Hand is free of swelling but some remains in forearm.

patient is up and about, however, it tends to reaccumulate. To prevent this, an elastic sleeve has been devised (Figs. 4 and 9). This sleeve is fitted snugly around the patient's arm early in the morning after the fluid has been drained out during the night. Heavy elastic material is used, similar to that employed in effective elastic stockings. Light nylon materials are not satisfactory. The sleeve extends from the distal portion of the palm to the midbrachium. Patients are instructed to don the elastic sleeve upon arising and to wear it throughout the day. After approximately 6 months of this treatment, some patients have been able to discard the sleeve without a recurrence of edema. One patient (Case 1) required in addition a tight-fitting rubber surgeon's glove to keep the edema out of her fingers. After a month of this treatment, she was able to abandon the glove without return of edema.

MASSAGE

Efflavage in the direction of lymph flow seems to be a logical procedure. Patients are instructed to massage their arms for at least 10 minutes twice a day, starting with short strokes at the shoulder and gradually increasing the length of the strokes to include even the fingers, "kneading out" the edema with each stroke. Whirlpool baths which afford gentle massage plus water pressure graded with maximum pressure peripherally, thus encouraging lymph and venous flow, have also been employed as an adjunct.

DEHYDRATION

At the onset of treatment, rapid subsidence of swelling can be hastened by dehydration. The patient is instructed to maintain a 2 gram salt diet. Mercurial diuretics are administered daily or every other day. This regimen is maintained only for the first week.

CASE REPORTS

CASE 1. Mrs. M. J., NYH No. 428954, aged 65 years (Figs. 3, 4, 5). Left radical mastectomy was performed in November, 1945 for canalicular scirrhous carcinoma of left breast with no discoverable axillary metastases. Swelling of the arm developed following operation. It commenced first in the brachium. Slowly through the course of the next 3 years the swelling involved the lower arm, wrist, and hand. The swelling usually subsided when the patient elevated her arm on a pillow, but during the 2 months immediately preceding vascular consultation, the swelling apparently became more fixed.

The patient was seen by the author on July 8, 1948. She was a tiny woman and was very apprehensive and disturbed about the appearance of her swollen hand. There was a large elliptical surgical scar across the left anterior thorax extending into the axilla. The axilla was deep; no nodes were palp-

TABLE I.—CASE 1

	Circumference in cm.		
	July 8, 1948	July 28, 1948	Dec. 16, 1950
Hand—above metacarpal bulge	18.5	18.1	18.0
Wrist	15.0	14.7	14.7
Maximum forearm	25.0	20.0	19.7
Maximum midbrachium	27.0	22.0	21.0

able. Oscillometric readings were normal. The entire arm was swollen. The brachium and lower arm were indurated, and the swelling did not pit, although the hand and fingers had a soft edema which pitted readily. Measurements are shown in Table I. Treatment consisting of nightly elevation, efflavage, and the use of an elastic sleeve and a snugly fitting rubber surgeon's glove was started at once.

Four weeks later the swelling was entirely cleared in the fingers, and hand. There was marked reduction in the forearm and arm. The rubber glove was discarded. After 6 months, the elastic sleeve was also eliminated. On the patient's last visit, 2½ years after beginning treatment, only a slight thickening remained in the forearm and arm. She continues to sleep with the arm elevated.

CASE 2. Mrs. H. M., Doctor's Hospital No. 79216, aged 55 years. A radical breast amputation was performed for adenocarcinoma in November, 1949. Following operation, a series of deep x-ray treatments was administered. Swelling developed first in the left shoulder area, then spread to the arm, forearm, and hand. The patient was admitted for treatment in June, 1950. She was placed in bed, and the arm suspended in elevation with counterweight traction. A 2 gram salt diet was prescribed, and the patient was given daily injections of 2 cubic centimeters of mercuhydrin intramuscularly. In addition she was given daily massage and whirlpool.

The swelling cleared rapidly. At the end of 1 week, the fingers, hand, and wrist were free of edema, and there was marked diminution in swelling of the forearm and arm. The patient was discharged with an elastic sleeve and instructed to continue elevation at night and self-massage. Two months later, there was further reduction in the swelling but some residual thickness remained in the upper arm.

TABLE II.—CASE 2

	Circumference in cm.			
	June 25, 1950	July 1, 1950	Aug. 30, 1950	April 17, 1951
Hand—above metacarpal bulge	18.7	18.2	18.2	18.2
Wrist	18.2	15.8	15.5	14.9
Maximum forearm	31.5	28.2	26.9	25.2
Maximum midbrachium	39.0	37.0	36.0	30.5

TABLE III.—CASE 3

	Circumference in cm.		
	June 20, 1950	July 1, 1950	Oct. 11, 1950
Hand—above metacarpal bulge	19.2	18.0	18.0
Wrist	18.4	13.5	13.5
Maximum forearm	30.4	25.2	23.4
Maximum midbrachium	36.2	28.5	26.5

CASE 3. Miss C. R., NYH No. 574001, aged 70 years. The patient had had a left radical mastectomy in 1925. She had received deep x-ray therapy before and after surgery. Edema developed in the arm descending to the hand and fingers. The swelling was worse after patient used her hands for a long period of time, such as in knitting. She had had two severe episodes of cellulitis which had responded to elevation and hot packs. Three weeks before admission the patient developed an infected thumb following a cut from opening a can. An intense, red, inflammation developed in the hand, forearm, and arm extending to the shoulder.

The patient was admitted to New York Hospital on June 20, 1950. The arm was suspended in countertraction; hot moist packs were applied; and antibiotics were given. The patient ran a septic fever for 2 days. Then the inflammation abated rapidly, and the patient was discharged 10 days later with an elastic sleeve, and instructions to sleep with the arm in elevation and to give herself massage.

A follow-up visit 3½ months later found the hand and wrist normal and the forearm and arm greatly reduced in size. According to the patient, her arm was smaller than it had been "in twenty years."

CASE 4. Miss M. M., Doctor's Hospital No. 81239, aged 54 years (Fig. 2). Patient had a left radical mastectomy at Memorial Hospital on September 4, 1945, Clinic No. 77A. She received 22 deep x-ray treatments after operation. The arm developed slight swelling in the brachium and forearm. In July, 1950, the swelling extended to the hand and fingers.

Patient was admitted to the hospital for 1 week. The arm was suspended in traction; hot moist packs were applied; a 2 gram salt diet was prescribed, and the patient received daily injections of mercurial diuretics, massage, and whirlpool. The swelling subsided rapidly. The patient was discharged with instructions to sleep with the arm suspended in traction, to give herself daily massage, and to wear a fitted elastic sleeve.

On a follow-up visit 1 month later, the fingers and hand were normal in size. Some residual swelling remains in the forearm and arm.

TABLE IV.—CASE 4

	Circumference in cm.		
	Oct. 21, 1950	Oct. 30, 1950	Nov. 28, 1950
Hand—above metacarpal bulge	22.8	18.5	18.5
Wrist	17.8	15.4	15.4
Maximum forearm	31.2	26.8	26.8
Maximum brachium	37.0	32.8	32.6

CASE 5. Miss M. B., NYH No. 553402, aged 53 years (Figs. 6 and 7). A left radical breast amputation was performed for carcinoma on October 14, 1949. One month later swelling developed in the arm. The area adjacent to the elbow became stiff and hard. In May, 1950, the hand and fingers became swollen.

The patient appeared for vascular consultation on May 26, 1950. At that time the only abnormality was the surgical incision and the swollen arm. For a distance of 10 centimeters above and below the elbow there was a woody, dense edema. It was pale and firm. The upper arm was swollen but soft, while a very soft edema was present over the forearm, hand, and fingers. Oscillometric studies were normal.

The patient was not hospitalized. She was instructed to suspend the arm in elevation at night, wear an elastic sleeve, and massage the arm daily. The swelling left the fingers, hand, and wrist rapidly. It has slowly subsided elsewhere. On the patient's last visit, October 18, 1950, the area around the elbow was soft.

CASE 6. Mrs. R. Q., NYH No. 2248, aged 55 years. This clinic patient was treated at New York Hospital Vascular Disease Clinic by our colleague, Dr. Burke. In 1940, a right radical mastectomy had been performed for carcinoma followed by a course of 15 deep x-ray treatments. The patient's arm commenced to swell. The swelling gradually increased in size through the years. In 1946, the patient was admitted for a gynecological operation. At that time it was noted that her right arm was "twice normal size." The swelling extended to the hand and fingers. She was seen in the Vascular Clinic in September, 1949, and instructed to elevate the arm, wear an elastic sleeve, give herself massage, and take whirlpool baths.

In 3 weeks the swelling had disappeared from the hand and was reduced in the forearm and arm. The patient was last seen on December 15, 1950. There was further reduction in swelling. She continues to wear her sleeve.

CASE 7. Captain M. W. (Army nurse), aged 41 years. In July, 1950, a right radical mastectomy was performed at Walter Reed Hospital for carcinoma. This was followed by 27 deep x-ray treatments totalling 5488 roentgens. Three months later, the arm commenced to swell. The swelling involved the shoulder, brachium, and forearm. The hand was only slightly swollen.

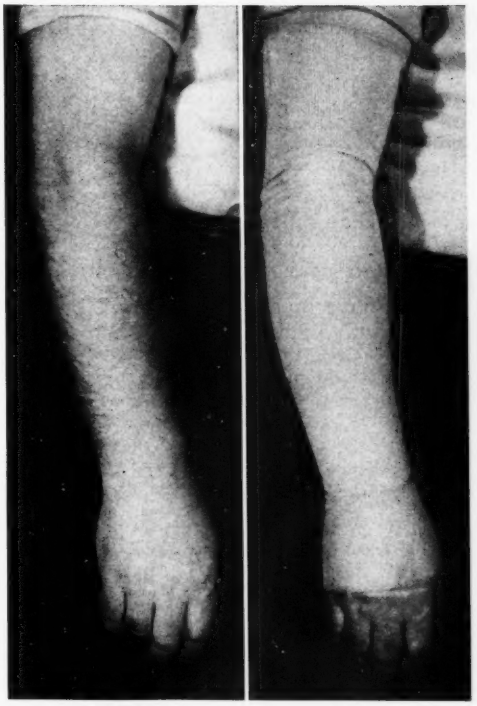

Fig. 8. Fig. 9.

Fig. 8. Case 8. Axillary vein thrombosis. The hand is swollen and cyanotic.

Fig. 9. Case 8. Elastic sleeve in place.

The patient was instructed to sleep with her arm suspended in traction, have an elastic sleeve made, take massage and whirlpool baths, and have mercurial diuretic injections every other day for one week while on a low salt diet. Swelling subsided quickly in the hand and forearm. Some edema remained in the upper arm 3 months later, but this area had become much softer.

TABLE V.—CASE 5

	Circumference in cm.	
	May 26, 1950	Oct. 18, 1950
Hand—above metacarpal bulge	18.5	17.0
Wrist	15.5	15.0
Maximum forearm	27.0	25.0
Maximum brachium	28.4	28.0

TABLE VI.—CASE 7

	Circumference in cm.	
	Dec. 16, 1950	Mar. 14, 1951
Hand—above metacarpal bulge	18.2	17.8
Wrist	16.0	16.0
Maximum forearm	26.2	24.1
Maximum brachium	33.2	31.1

AXILLARY VEIN THROMBOSIS

In a similar manner we have treated 6 patients with axillary vein thromboses which were not associated with surgery. A typical example is presented in the following case report:

CASE 8. Mr. V., aged 42 years (Figs. 8, 9). Patient was a laborer in a rug factory. On April 23, 1945, he tripped over a "worsted beam" and fell on his right shoulder. His arm was splinted for 5 weeks. When the splint was removed, it was noted that the arm was blue and swollen. The arm remained painful and swollen. The patient was unable to return to work. When he was seen by Dr. Irving Wright on December 17, 1946, the arm and hand were swollen and cyanotic. There was increased pressure in the veins of the right hand as compared to those in the left hand. The veins in the left hand emptied when the hand was raised to the level of the auricles, while those of the right hand required further elevation of approximately 5 inches. Our diagnosis in this case was axillary venous thrombosis.

The patient was instructed to elevate the arm at night and to wear an elastic sleeve during the day. On his next visit in March, 1947, there was great improvement. The swelling had disappeared but cyanosis persisted. The patient had resumed work, but continued to wear the elastic sleeve as it gave him a feeling of support.

SUMMARY AND CONCLUSIONS

Swelling of the arm secondary to axillary surgery and to axillary venous thrombosis is a disabling condition. It has not been easily amenable to surgical or medical treatment. The application of physiological principles resulted in the development of a method of treatment employing gravity, compression, massage, and dehydration. Marked improvement in swelling, pain, and cosmetic appearance has resulted in a small group of patients so treated. This improvement has been maintained.

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CHRONIC THYROIDITIS

Supravital Studies of Surgical Goiter Specimens

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IN a review of 187 cases of chronic thyroiditis encountered in the Lahey Clinic, Marshall, Meissner and Smith state that the confusion and disagreement of surgeons regarding chronic thyroiditis is due to the difficult classification of this disease and to a poorly defined terminology.

The three forms of chronic thyroiditis which are distinguished in modern treatises (1, 2) are attached to three names: Riedel, a German surgeon who in 1896 described the iron-hard fibroplastic goiter involving also the surrounding tissue, generally known as Riedel's struma; Hashimoto, a Japanese surgeon who 16 years later gave the characteristics of the struma lymphomatosa with diffuse and follicular lymphocytic hyperplasia (while this form is appropriately named "lymphadenoid goiter" by Williamson and Pearse, most surgeons call it Hashimoto's disease); third, the name of DeQuervain, a Swiss surgeon who in 1940 described the subacute giant-cell thyroiditis. This type is characterized by a subacute inflammatory process with foreign body giant cells and formation of pseudotubercles.

The clinical diagnosis of chronic thyroiditis is difficult—a correct preoperative diagnosis in the 187 patients seen at the Lahey Clinic was made only in 44 patients, or 23.5 per cent. The hardness of the enlarged gland may suggest a malignant form of goiter. Histological study of the surgical specimen, or needle biopsy, is as a rule necessary to classify the disease correctly.

Our experience with the incidence of chronic thyroiditis in surgical goiter material is the same as that of the Lahey Clinic, namely that chronic thyroiditis is encountered fairly frequently in thyroid clinics. During the last 10 years we saw 169 cases of chronic thyroiditis among 2,031 patients requiring thyroidectomy. This represents an incidence of 8.3 per cent. In our survey, no cases are included occurring in exophthalmic goiter, solitary adenoma, or multinodular colloid goiter, although lymphocytic infiltration, with or without formation of lymph follicles, may frequently complicate such already diseased glands.

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Dividing our 169 cases of chronic thyroiditis into the three groups, we had 8 cases of Riedel's struma (fibroplastic form), 146 cases of Hashimoto's disease (lymphadenoid goiter), and 9 cases of giant cell thyroiditis of DeQuervain, while 6 could not be classified and represented earliest stages of chronic thyroiditis.

The clinical and histological characteristics of chronic thyroiditis have been exhaustively described by recent investigators (Wegelin, Joll, Lee, Marshall and coworkers, Crile, Schilling, and Graham). We would like therefore to present the results of our supravital studies which lead to a new conception of the etiology of chronic thyroiditis.

MATERIAL AND METHOD

The surgical specimens were brought to the laboratory immediately after removal. The fresh tissue was sliced in several frontal sections and from gray foci in the diffuse tissue small particles, 1 to 2 millimeters in diameter, were transferred with curved scissors to a warm slide. With dissecting needles the tissue was teased into fine shreds and covered with a solution of neutral-red in saline, 1:10,000, by medicine dropper. After a cover glass was applied, the slide was placed in a large Petri dish, the bottom of which was covered with a wet piece of filter paper. The closed Petri dish was kept at 37 degrees C. for 30 minutes and then the tissue was examined under the high dry power of the microscope. As a rule, three slides were prepared from each of the goiter specimens.

TABLE I.—INTRAFOLLICULAR AND INTERSTITIAL MACROPHAGES STAINED WITH NEUTRAL-RED IN SUPRAVITAL PREPARATIONS

No.	Sex	Age	Histological type of goiter	F*	S†	PBI‡
1	M	70	Colloid adenoma		x	4.2
2	F	51	Cystic colloid adenoma		x	4.8
3	F	34	Exophthalmic goiter	x		
4	F	38	Colloid adenoma		x	6.2
5	F	35	Colloid adenoma		x	7.5
6	F	55	Nodular colloid goiter	o	o	14.0
7	F	32	Diffuse colloid goiter, thyroiditis	x		
8	F	42	Exophthalmic goiter	xx	x	11.0
9	F	51	Nodular colloid goiter		x	14.0
10	F	32	Exophthalmic goiter	x		7.0
11	F	34	Nodular colloid goiter	x	x	
12	F	68	Nodular colloid goiter	o	o	
13	F	35	Exophthalmic goiter	x		16.5
14	F	22	Diffuse colloid goiter, thyroiditis	x	x	
15	F	56	Nodular colloid goiter		x	4.0
16	F	36	Hemorrhagic cyst			3.4
17	M	41	Fetal adenoma			5.1
18	F	58	Hürthle cell adenoma			
19	F	48	Colloid adenoma	x		5.4
20	F	28	Diffuse colloid goiter			20.0
21	F	45	Exophthalmic goiter	x		
22	F	57	Nodular colloid goiter	x		
23	F	66	Multiple colloid adenomas	x	x	
24	F	52	Multinodular colloid goiter	x		5.0
25	F	26	Colloid adenoma		x	
26	F	40	Diffuse colloid goiter, thyroiditis	x		
27	F	44	Colloid adenoma	x	x	6.5
28	F	70	Adenocarcinoma	x		
29	F	65	Multiple colloid adenomas	x		4.8
30	F	48	Colloid adenoma	x		5.8
31	F	53	Colloid adenoma	x		
32	F	51	Hürthle cell tumor	x		5.2
33	F	57	Colloid adenoma		x	12.2
34	M	63	Colloid and fetal adenoma			12.2
35	F	53	Lymphadenoid goiter	x	x	4.6
36	F	39	Colloid adenoma			8.2
37	F	21	Exophthalmic goiter			
38	F	38	Fetal adenoma		x	6.2
39	F	45	Exophthalmic goiter	x		15.5
40	F	50	Diffuse colloid goiter	x		18.0
41	F	34	Exophthalmic goiter	x		14.5
42	F	55	Nodular colloid goiter	x		6.0
43	F	48	Lymphadenoid goiter			
44	F	58	Nodular colloid goiter	x	x	7.5
45	F	37	Colloid adenoma	x		
46	F	40	Nodular colloid goiter			
47	F	63	Nodular colloid goiter			6.0
48	F	59	Colloid adenoma	x	x	
49	F	48	Colloid adenoma			
50	F	35	Exophthalmic goiter	x	x	14.0
51	F	47	Colloid adenoma			
52	F	64	Colloid adenoma	x		
53	F	62	Nodular colloid goiter	x		12.0
54	M	23	Adenocarcinoma			5.0

*Intrafollicular macrophages
†Interstitial macrophages
‡Protein bound iodine of serum in mgm. per cent

RESULTS

Table I shows the findings of our supravital studies.

In most of the 54 goiters examined we found large cells containing coarse salmon-red granules in their cytoplasm. In 30 specimens these large cells, which had taken up neutral-red, were found within the lumen of follicles; in 18 goiters stained cells were seen in the interfollicular tissue.

There could be no doubt about the nature of these cells stained supravitally with neutral-red. They were macrophages. The significance of these findings becomes apparent if we remember the studies on thyroids of experimental animals. The first to describe invasion and ingestion of colloid by macrophages (colloidophagy) were Leo Loeb and Gray (10). Thurston, after injection of pituitary extract, saw phagocytosis of colloid in the thyroid of guinea pigs, rats, and pigeons.

According to Eggert, phagocytes play an important role in the resorption of colloid. After giving large doses of thyrotropic hormone he saw, in the thyroid of lizards, wandering cells enter hypertrophic follicles, ingest colloid, and carry it into the interfollicular vessels.

R. G. Williams studied living thyroid follicles in transparent chambers inserted in rabbits' ears. In the colloid of some activated follicles he saw from 1 to 20 wandering cells. Their number changed from day to day in the same follicles, eventually disappearing. Thyrotropic hormone increased the activity of these colloidophages; there was no evidence of degeneration of the epithelium in the invaded follicles. In human thyroids, this process of colloidophagy has never been studied.

While cells in follicular lumens have been described repeatedly, they were regarded as desquamated, degenerated cells of the follicle wall.

To learn about the significance of the intrafollicular macrophages found in our supravital preparations, we studied paraffin sections of thyroids showing chronic inflammation. In 63.9 per cent of 435 goiters removed by operation and in 16.2 per cent of 619 thyroids obtained by autopsy we found foci of chronic inflammation characterized by aggregation of

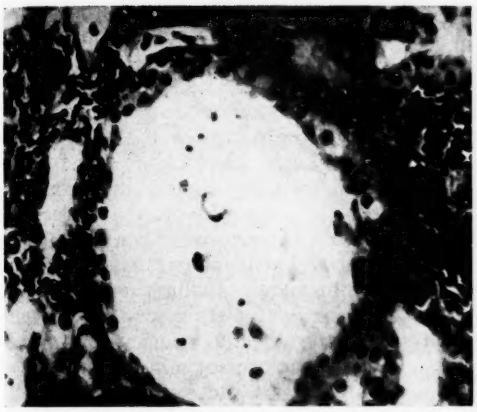

Fig. 1. Intrafollicular monocytes (macrophages) in chronic thyroiditis.

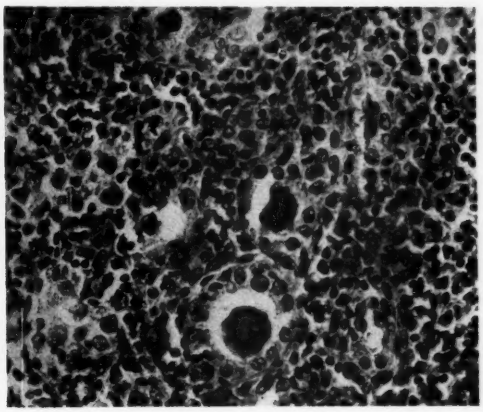

Fig. 2. Resorption of colloid by monocytes. (Second stage of colloidophagy.)

lymphocytes. Near the groups of lymphocytes, almost without exception, follicles were noticed which contained in their lumen large mononuclear cells. These intrafollicular cells varied in number from 1 to 20 in one lumen. They were of large size, their cytoplasm was eosinophilic, and their nucleus was oval or kidney-shaped. They did not resemble thyroid cells at all, and there was no evidence of degeneration of the involved follicles. Therefore the view of most pathologists that these are desquamated epithelial cells can be refuted. Our supravital studies establish their nature as macrophages.

All writers agree that the cause of chronic thyroiditis has never been satisfactorily explained. Chemical, bacterial, or viral agents responsible for this disease have never been found. We believe that the invasion and ingestion by macrophages as seen by us offers a better understanding of the cause of chronic thyroiditis.

Two questions arise if one tries to associate colloidophagy with chronic thyroiditis: (1) What causes this invasion of intrafollicular colloid by wandering cells? From animal experiments we know that an excess of thyrotropic hormone stimulates colloidophagy in

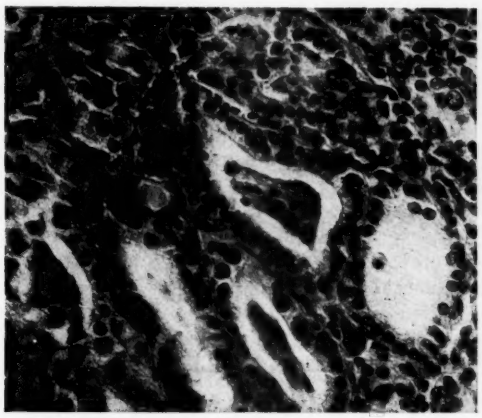

Fig. 3. Formation of intrafollicular syncytium by fusion of monocytes.

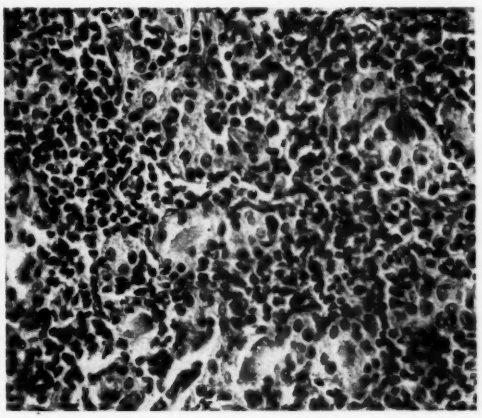

Fig. 4. Colloid remnants in interstitial tissue attracting lymphocytes.

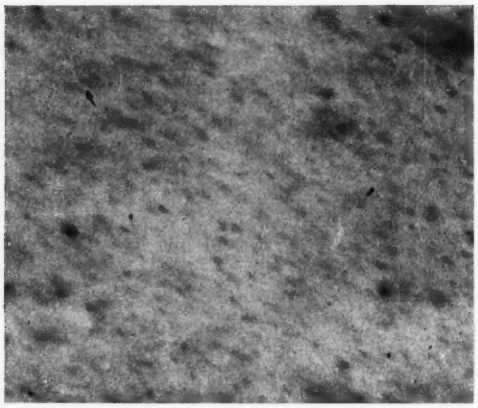

Fig. 5. Electronmicrograph (magnification, 30,000) of colloid from normal thyroid.

the normal thyroid. Lymphadenoid goiter, in which colloidophagy with aggregation of lymphocytes is the outstanding characteristic, has been related to an excess of thyrotropic hormone. We feel justified, therefore, in assuming that an excess of thyrotropic hormone or increased sensitivity of the thyroid cell to thyrotropic hormone changes the properties of the colloid in such a way that it attracts macrophages. Electron microscopic studies seem to point in the same direction. The extract of lymphadenoid goiters and of rabbit thyroids activated by thyrotropic hormone looks different in the electron microscope from that of normal thyroids. While the colloid of the normal thyroid gland appears in the electron microscope to be composed of globules of about 25 mμ in diameter, the colloid of lymphadenoid goiter and of rabbit thyroids stimulated with thyrotropic hormone reveals bizarre, wedge-shaped, angular and sharp-edged particles measuring about 160 mμ in length. These structural changes may well indicate chemical alterations of the colloid in chronic thyroiditis, which would explain the attraction of macrophages by chemotaxis.

The second question which concerns us has to do with the lymphocytic infiltration, often associated with formation of lymph follicles, in chronic thyroiditis. From the study of supravital and paraffin preparations of thyroids from various stages of chronic thyroiditis, one can reconstruct the following sequence of events: Three stages of colloidophagy can be distinguished. In the earliest stage (Fig. 1), one or several macrophages are seen in the colloid of a follicle, and often a small gap between cells of the follicle wall shows where they entered the follicle lumen. The follicle wall does not show any signs of degeneration in this stage. In these involved follicles, no difference from the normal colloid, color, or consistency could be demonstrated in paraffin section by histologic staining methods, in frozen sections by fat stain, or in polarized light. Only the already mentioned electron micrographs at 30,000 magnification show a difference in the size and form of the colloid particles. In a later stage (Fig. 2) the colloid in the involved follicles disappears more and more and the macrophages become larger and stain intensively with eosin. This can be interpreted only as ingestion of the colloid by macrophages.

Figure 3 illustrates the second stage of colloidophagy. The follicles have become collapsed, the form of the lumen is oval, triangular, or slit-like, and the epithelial cells of the follicle are higher than the cells of noninvolved follicles. The colloid has entirely disappeared and in the lumen is a syncytial mass of cells, apparently macrophages, which have fused together. Since the nuclei appear pyknotic, we believe these cells are not viable.

From animal experiments it is known that the macrophages, after ingestion of colloid, leave the follicle lumen and re-enter the circulation. From supravital studies we believe that this happens also in human thyroids. We have often seen macrophages with coarse red granules in the interfollicular septa. Some of these cells apparently are unable to re-enter the circulation, but are so loaded with colloid that they become stranded in the interfollicular stroma. Here they degenerate and the liberated colloid attracts lymphocytes by chemotaxis. The latter form a wall around the colloid plaques and often lymph follicles with germinal centers are formed. In Figure 4 we see irregular masses of colloid in the center of a germinal center.

The colloid, if injected into connective tissue, attracts lymphocytes and macrophages

by chemotaxis as demonstrated by Ferguson in experiments on guinea pigs. By injection of colloid into the subcutaneous tissue, he produced a severe foreign body reaction with numerous giant cells and fibroblasts. Most writers believe that chronic thyroiditis begins with degeneration of thyroid epithelium caused by a virus or bacterial agent, and that the lymphocytes are attracted by products of protein-decomposition of the cells. While Crile (1, 2) and others describe foreign body giant cells engulfing colloid, they state that the colloid is "spilled" into the tissue by necrosis of follicle epithelium. Our observations, on the contrary, show that macrophages enter the follicle lumen which is lined by completely normal cells, that they ingest colloid and transport it to the interfollicular stroma. Here they degenerate and the liberated colloid attracts lymphocytes and gives rise to the formation of lymph follicles. Apparently the chemical constitution of the colloid has become altered in the cytoplasm of the macrophages and the liberated colloid now contains substances which attract more lymphocytes than macrophages. From the studies of Sabin on fractionated constituents of the tubercle bacillus, it seems that macrophages are attracted by lipid fractions and lymphocytes by carbohydrate fractions of the tubercle bacillus. Similar substances may be responsible for the attraction of macrophages of the intrafollicular colloid and of lymphocytes of the extrafollicular colloid deposited by macrophages in the stroma.

The degeneration of follicle epithelium in the later stages of thyroiditis which we have also seen can be easily explained as due to accumulation of lymphocytes around and between follicles. The latter become isolated by the lymphocytes from the nourishing and stimulating influence of the blood, resulting in atrophy from disuse and lack of nourishment, leading finally to degeneration. The formation of fibrous tissue, which is so striking in the fibroplastic type of chronic thyroiditis (Riedel's struma), is the final act which occurs following the destruction of the thyroid epithelium.

Our view of the histogenesis of chronic thyroiditis is supported by animal experiments.

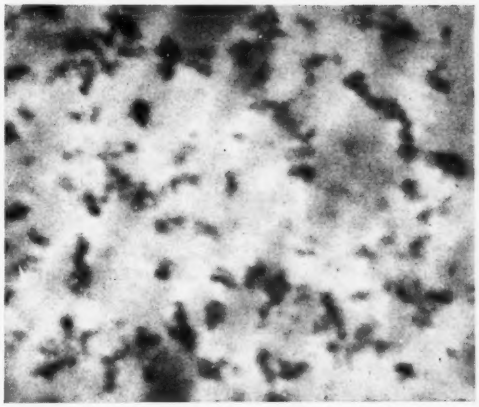

Fig. 6. Electronmicrograph (magnification 30,000) of colloid from chronic thyroiditis.

While the full established picture of chronic thyroiditis has not been produced in animals, masses of lymphocytes have been described by Thurston in thyroids of animals after injection of thyrotropic hormone.

CONCLUSIONS

1. During the last 10 years, 169 cases of chronic thyroiditis were encountered among 2,031 patients requiring thyroidectomy (8.3%). Small foci of chronic thyroiditis characterized by aggregations of lymphocytes are common in exophthalmic goiter and nodular goiter (63.9%) and not infrequent in normal sized thyroids obtained by autopsy (16.2%).

2. There are only quantitative differences between the severe grades of chronic thyroiditis (Riedel's, Hashimoto's, and DeQuervain's type) and localized foci of chronic thyroiditis in exophthalmic goiter and nodular goiter. Both show the same morphologic characteristics, the only difference being in intensity.

3. There is much disagreement regarding the etiology of chronic thyroiditis. All attempts to find bacteria or a virus as the cause of chronic thyroiditis have been unsuccessful.

4. Supravital studies of fresh surgical material suggest a new concept of the cause of this disease. By change in the composition of colloid, macrophages are attracted which invade the lumen of follicles, ingest colloid, and return into the interfollicular stroma. If they

disintegrate in the stroma the liberated colloid attracts lymphocytes which aggregate around the colloid and produce the anatomical picture of chronic thyroiditis. As cause of the colloid changes responsible for attraction of macrophages, excess of thyrotropic hormone seems the most important factor.

5. Our new concept of chronic thyroiditis as a chemical inflammation—comparable to lipogranuloma—is supported by experimental, supravital, and electronmicroscopic findings.

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EVALUATION OF PREOPERATIVE SKIN PREPARATION

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ALTHOUGH there is general agreement regarding the qualities essential to a good antiseptic, the methods used to evaluate these agents have been largely inadequate. Chemical differences between compounds and differences in modes of action make it impossible to find one test which is universally applicable and reliable. It has been recommended that an antiseptic be subjected to a number of tests, each evaluating one or more different properties; the so called "profile evaluation" (12).

There are in general three types of tests. The first, or in vitro type of test, has many variations. The variations include the F.D.A. phenol coefficient method (7), the total swirl test (8), the many plate assay methods, the contaminated blade technique of Spaulding (16), and others. These are limited in their clinical applicability, but serve a useful purpose in screening large numbers of new compounds, and in determining broad spectra of antibacterial activity.

The second type of test yields an antibacterial activity/tissue toxicity ratio. These range from the early experiments of Russell on the effect of gentian violet on protozoa and frog tissue to the chick embryo and animal techniques of more recent times (1, 2, 3, 4, 13, 14, 15, 17).

Clinical studies make up the third type of test. The bacteriologic study of segments of skin excised or scraped after the application of antiseptics has been reported for more than 60 years (10).

The effectiveness of a number of antiseptics used in the preoperative preparation of the patients' skin was compared by means of a group of four experiments. These include an in vitro study, a modification of the chick embryo tests, animal experiments, and a clinical study.

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IN VITRO STUDIES

A modification of the "total swirl" test as recommended and described by Ostrolenk was used to determine the action of the agents studied against some of the bacteria most commonly encountered on skin. This test was found to be the most reliable of the in vitro tests by Ostrolenk in a series of experiments in which he compared five commonly used methods, using eight compounds and four types of organisms.

The cultures used were of organisms recently isolated from patients' skin, transferred daily to 10 cubic centimeters of fresh medium, a 4 millimeter loop being used. One-tenth cubic centimeter of each test culture was added to 1.0 cubic centimeter of each antiseptic and the mixture was agitated on a mechanical shaker for 5 minutes. Forty cubic centimeters of thioglycollate broth was then poured on top of the mixture, and the tube incubated for 4 days at 37 degrees C. A series of 10 such tests was run in evaluating the action of each of 8 antiseptics against each of 8 organisms.

The organisms tested were Bacillus coli, Bacillus subtilis, Bacillis proteus, Bacillus pseudomonas, diphtheroids, hemolytic Staphylococcus albus, hemolytic Staphylococcus aureus, and hemolytic Streptococcus.

The antiseptics tested were zephiran (1:1,000), both alcoholic and aqueous solutions, tincture of acrizane[1] (1:200), iodine tincture, *p*Hisohex (*p*Hisoderm with 3 per cent hexachlorophene), ethyl alcohol 70 per cent, ethyl ether, and a solution of chrysoidin Y, 2 per cent (W/V) in 99 per cent isopropyl alcohol. The latter has been used as a preoperative

[1]Acrizane is a more recently introduced antiseptic, still under experimental observation, whose formula is 9-parahexyl oxyphenol-9-chloro-10-methyl acridine.

TABLE I.—EFFICIENCY OF ANTISEPTICS IN VIVO (TOTAL SWIRL TEST)
PER CENT NEGATIVE CULTURES

Organism	Acrizane (Tr. 1:200)	Zephiran (alcoholic)	*p*Hisohex (3% G-11)	Zephiran (aqueous 1:1000)	Antiseptic iodine (Tr. 2%)	Ethyl alcohol (70%)	Ether	Chrysoidin Y (99% isopropyl alcohol)
Bacillus proteus	100	100	60	40	100	0	80	0
Bacillus pseudomonas	100	100	100	70	100	100	100	100
Bacillus subtilis	100	100	100	100	40	100	90	90
Hemolytic streptococcus	100	100	100	100	100	40	30	0
Hemolytic Streptococcus albus	100	100	100	100	80	80	90	60
Hemolytic Staphylococcus aureus	100	100	100	100	80	70	0	0
Diphtheroids	100	100	100	100	100	50	50	0
Bacillus coli	100	100	100	100	100	60	0	0

paint at the University Hospital for several years.

In Table I are tabulated the results of these experiments with efficiency of the antiseptic expressed as percentage of tubes showing complete inhibition of bacterial growth at end of test period. Tincture of acrizane 1:200, alcoholic zephiran, and *p*Hisohex are most effective.

CHICK EMBRYO STUDIES

A modification of the chick embryo technique of Green and Birkeland was used to compare antibacterial activity with tissue toxicity of the antiseptics under consideration.

Technique. Eleven day old incubated chick eggs were candled to ascertain viability, and the position of the air space and embryo

TABLE II.—EFFECT OF AGENTS USED IN SKIN PREPARATION ON STAPHYLOCOCCAL INFECTIONS OF CHORIOALLANTOIC MEMBRANE OF CHICK EMBRYO

Solution tested	Dilution (in sterile distilled water)	Number embryos tested	Number embryos surviving	No. survivors showing each degree of infection			
				o	+	++	+++
Tincture of acrizane	1:1000	30	8	3	4	1	
	1:5000	30	19	2	6	7	4
	1:10,000	30	27	2	5	11	9
Zephiran in (70% ethyl alcohol)	1:5000	30	17	4	2	6	5
	1:10,000	30	18	3	3	9	3
Zephiran (aqueous)	1:1000	30	11	4	3	2	2
	1:5000	30	18		3	3	12
*p*Hisoderm with hexachlorophene 3% (*p*Hisohex)	1:1000	30	19	2	3	3	11
	1:5000	30	13	2		4	7
	1:10,000	30	12				12
Tincture of iodine U.S.P. 2%	1:5000	30	13			4	9
	1:10,000	30	15			3	12
Ethyl alcohol 70%	1:5000	30	14			2	12
	1:10,000	30	22			5	17
Chrysoidin Y (2% in 99% isopropyl alcohol)	1:1000	30	19			3	16
	1:5000	30	17				17
Controls Infected and treated with sterile 0.9% NaCl solution		32	18				18
Not infected and treated with sterile 0.9% NaCl		39	35	35			

o—No. organisms recovered +—1 to 10 colonies ++—More than 10 colonies, not overgrown +++—Overgrown

marked on the shell. The shell was cleansed with 70 per cent ethyl alcohol and a small perforation made over the air space. At the site of the embryo a small slit was made through the shell to the shell membrane and a drop of sterile saline solution placed thereon. Pressure with the side of a No. 19 needle on the shell membrane allowed the saline to run between the shell and chorioallantoic membranes, facilitating displacement of the egg contents into the natural air space when suction was applied to the perforation over the air space.

With the egg lying on its side, 0.02 milliliter of a 1:100 dilution of a 24 hour culture of Staphylococcus aureus F.D.A. strain 209, was dropped on the chorioallantoic membrane through the slit by means of a tuberculin syringe and a half inch No. 27 needle. The slit was covered, but not tightly sealed, with scotch tape. The eggs were placed slit side up in an incubator kept at 100 to 101 degrees Fahrenheit. Eighteen to 24 hours later and daily thereafter for 4 days 0.2 milliliter of antiseptic solution was dropped on the infected membrane. The eggs were candled daily, and those which died before completion of the therapy were cultured to eliminate embryos dying from contaminants. Control experiments consisted of series of infected and un-

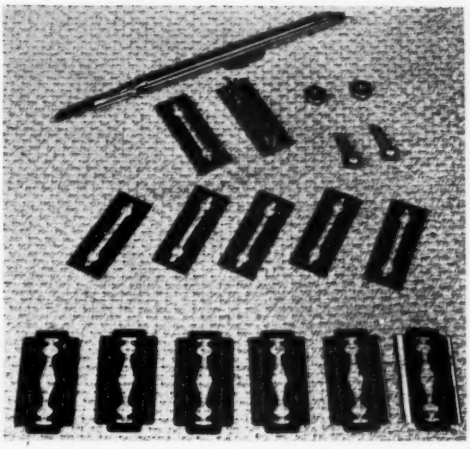

Fig. 1. Equipment—the scalpel with double blades, and the component parts of the razor block unassembled.

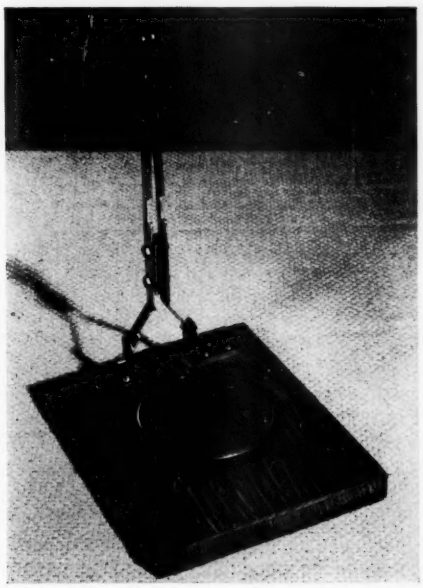

Fig. 2. The equipment used to cut uniform, small pieces of skin. The skin specimen is in the center of the cork lined Petri dish.

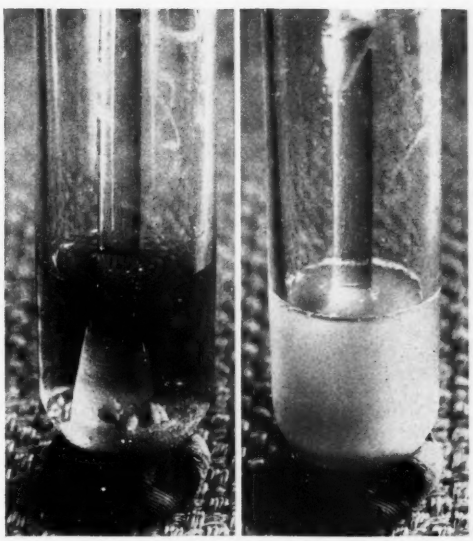

Fig. 3, left. Tube containing sand, pestle, nutrient broth, and skin segment before maceration.

Fig. 4. Tube containing sand, pestle, nutrient broth, and skin segment after maceration.

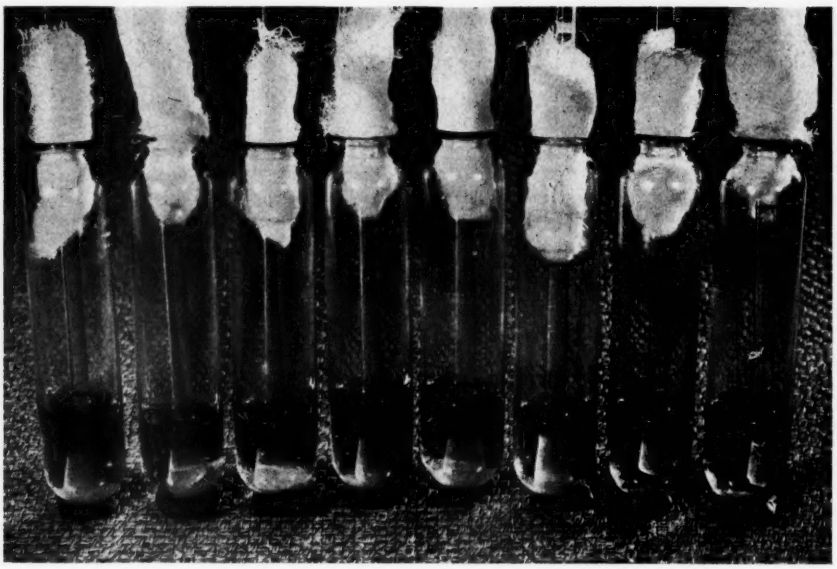

Fig. 5. A representative series of tubes containing nutrient broth, pestle, sea sand, and skin prior to incubation.

infected embryos, treated with sterile 0.9 per cent sodium chloride solution in place of the antiseptics.

On the day following the last treatment the chorioallantoic membrane was exposed by carefully removing the shell over it, and the membrane stroked with a moist cotton culture swab which was then streaked on half of a blood agar plate. Results were recorded in degrees of infection persisting on the membrane as evidenced by the number of colonies growing on the plate after 24 hours' incubation. The degree of infection was rated from 0 to 3 plus, as shown in Table II; 0 indicates complete inhibition of bacterial growth by the antiseptic, and 3 plus, complete failure to control bacterial growth.

A comparison of results obtained with several dilutions of six antiseptics is presented in Table II. The dilutions shown are absolute, with sterile distilled water as the diluent. Acrizane and alcoholic zephiran appear to be most effective, producing the highest antibacterial activity/tissue toxicity ratio. *p*HISOHEX and aqueous zephiran rated next. Iodine, ethyl alcohol, and chrysoidin Y solution were approximately equally ineffective in controlling the infection, with evidence of moderate toxicity.

ANIMAL STUDIES

Attempts at evaluation of agents and methods used in the preoperative preparation of skin by clinicobacteriological studies have been made from the earliest days of the aseptic era.

As described by Price and demonstrated by Lovell, there are two strata of bacterial flora in the skin. The first, called the transient flora, found in the superficial horny fat, crypts, and crevices, is the flora usually cultured by the methods used by many previous investigators who obtained their cultures from only the skin surface. This flora is obviously the most easily removed or inactivated by mechanical and chemical cleansing. The resident flora, located in the deeper crypts, hair follicles, and sebaceous glands, is probably seldom reached by antiseptics or attempts at culture.

Many authorities agree that the bacteria of this resident stratum are the ones most likely responsible for the contamination of so called

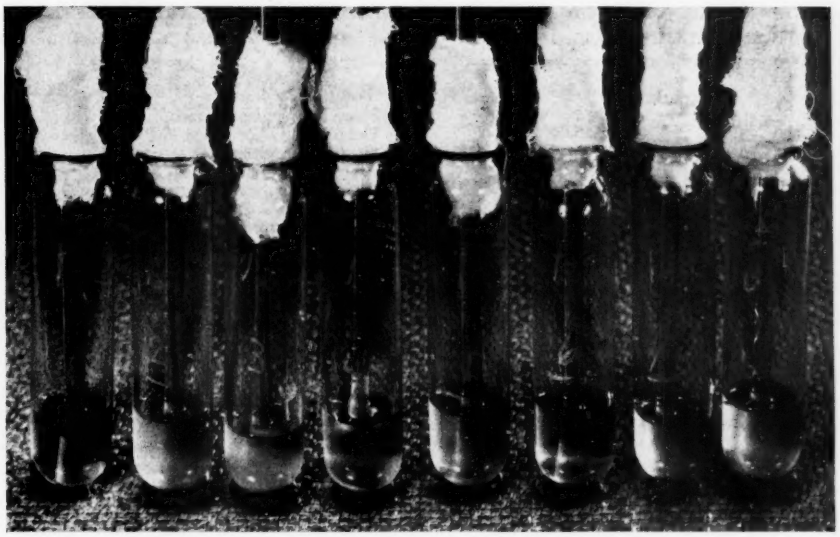

Fig. 6. A representative series of tubes containing macerated skin specimen after 72 hours' incubation. All tubes except number one and number six show evidence of bacterial growth.

'clean wounds," since during and after operation they may, under the cover of drapes and dressings, proliferate and rise to the surface, or they may be released along the line of incision by the scalpel (5, 6).

In the previous in vivo experiments using skin biopsies described in the literature there appear to be two major defects. First, when cultures were made of the full thickness of the skin, the lack of uniformity of the size of the skin specimens cultured was a serious fault. The second error may be described as a lack of adequate sampling in each individual subject.

In the experiments reported here, an attempt was made to obtain uniform and adequate samples from each subject, and each sample was treated in a fashion designed to provide complete and representative analysis.

In order to test some of the agents used for preoperative skin preparation against a known and varied bacterial flora, dogs were selected as subjects. The abdomen was shaved dry with electric clippers. The preparation being tested was applied to one half of the abdomen, the other half being used as a control. Intravenous nembutal anesthesia was used. Full thickness skin strips were taken from each side of the abdomen, by means of a double-bladed knife, the blades of which were fixed 2.0 millimeters apart (Fig. 1). The skin was separated grossly from the subcutaneous fat by trimming it off with a pair of scissors. Each skin strip was placed immediately in a sterile cork lined Petri plate, and 5 random segments were cut, each segment consisting of 5 pieces measuring 2.0 by 1.0 millimeter on the skin surface. A block of six sterile razor blades mounted 1.0 millimeter apart in a stainless steel handle was used to prepare these sections (Figs. 1 and 2). Each segment so prepared was transferred under sterile precautions to a thick-walled glass culture tube containing a glass pestle, sterile sea sand, and 15 cubic centimeters of brain-heart infusion broth. Figure 3 shows such a tube with the skin segment added. The skin was thoroughly macerated by means of the pestle and sand. Figure 4 shows a tube after maceration, the skin having been reduced to a fine suspension in the nutrient broth. This important step releases the bacteria contained in all strata of the skin. The tubes were incubated for 72 hours at 37 degrees centigrade. Figures 5 and 6 show a

TABLE III.—METHOD OF RATING BACTERIAL GROWTH FROM ANIMAL SKIN SECTIONS

Bacterial growth	Description	Antiseptic efficiency rating
o	No growth in 72 hours	4
*–	Retarded (growth in 72 hours)	3
*	Moderately retarded (growth in 48 hours)	2
**	Slightly retarded (growth in 24 hours)	1
***	Profuse growth in less than 24 hours	o

Thus five segments per skin strip times five animals per series times four points for complete bacteriostasis equals 100 per cent—highest possible rating.

series of tubes before and after incubation. Subcultures were made on tubes showing growth at 24 hour intervals, and at the end of the 72 hours on all tubes. A number of the tubes in which the broth remained clear at the end of 72 hours showed bacterial growth on subculture. Efficiency of the preparation is demonstrated by the degree of inhibition of bacterial growth from each of the skin sections. The method of rating antibacterial efficiency in the animal studies is shown in Table III. Five animals were used in evaluating each type of preparation. Table IV shows the results obtained using tincture of acrizane 1:200, tincture of iodine 2 per cent, tincture of merthiolate 1:1000, *p*HISOHEX (3 per cent hexachlorophene), chrysoidin Y 2 per cent (W/V) in 99 per cent isopropyl alcohol, and aqueous zephiran 1:1000. All were applied at the time of biopsy, and left on the skin for a period of time sufficient to drape the animal, not less than 2 nor more than 5 minutes, except in the case of the *p*HISOHEX, which was used to scrub the skin for 2 minutes 12 to 24 hours prior to biopsy, in addition to its use immediately preoperatively.

Tincture of acrizane and tincture of iodine proved to be most efficient in these animal studies. Table V lists the types of bacteria cultured from the skin of the dogs, and the frequency of their occurrence. Such a varied flora should provide as rigorous a test of an antiseptic as could be desired.

HUMAN STUDIES

The methods used in the animal experiments were applied to the human studies,

TABLE IV.—ANIMAL STUDIES EFFICIENCIES OF SKIN PREPARATIONS (FIVE DOGS IN EACH GROUP)

Preparation used	%Efficiency animal skin
Tincture of acrizane 1:200	84
Tincture of iodine 2%, U.S.P. XIV	67
Tincture of merthiolate 1:1000	54
*p*HISOHEX (*p*HISODERM with hexachlorophene 3%)	40
Chrysoidin Y solution	46
Zephiran (aqueous) 1:1000	36

with a few exceptions. No control or untreated specimens were taken from the patients' skin. Eight random segments were cut from each skin strip obtained from patients instead of five as used in the dogs. The skin biopsies were made from the chest, abdomen, or flanks. No less than five and no more than eight specimens in each series of patients was obtained from the inguinal region. This precaution was necessary because of the discovery that the skin from the inguinal region was uniformly more heavily contaminated than that from other parts of the chest and abdominal wall. Each preparation was used on 25 patients at the time of regularly scheduled operations. Each antiseptic agent was applied and left in contact with the skin for the length of time necessary to drape the patient, never less than 2 minutes or longer than 5 minutes before the skin specimen was obtained. The *p*HISOHEX preparation was used with scrubbing for 2 minutes 12 to 24 hours prior to operation in addition to its use as an immediate preoperative scrub. None of the other preparations were used prior to the time of the operation.

Eight types of skin preparation were tested. They were: tincture of acrizane 1:200, tincture of iodine 2 per cent in 95 per cent ethyl alcohol, allowed to dry, then removed with 70 per cent ethyl alcohol; ethyl ether followed by chrysoidin Y, 2 per cent (W/V) in 99 per cent isopropyl alcohol, *p*HISOHEX (*p*HISODERM with 3 per cent hexachlorophene), tincture of merthiolate 1:1000, aqueous zephiran 1:1000, chrysoidin Y 2 per cent (W/V) in 99 per cent isopropyl alcohol, and a "five cup" preparation consisting of successive applications of tincture of green soap, sterile water, ethyl alcohol 70 per cent (W/V), ethyl ether, and chrysoidin Y, 2 per cent (W/V) in 99 per cent isopropyl alcohol.

TABLE V.—TYPES AND FREQUENCY OF OCCURRENCE OF BACTERIA IN ANIMAL SKIN (AS FOUND IN BOTH CONTROL AND TREATED SPECIMENS)

Bacteria	Frequency
1. Micrococcus albus	26
2. Nonhemolytic streptococcus	24
3. Hemolytic Staphylococcus albus	17
4. Bacillus subtilis	15
5. Streptococcus viridans	14
6. Escherichia coli	11
7. Bacillus proteus	4
8. Hemolytic Staphylococcus aureus	3
9. Nonhemolytic clostridia	3
10. Micrococcus aureus	2
11. Hemolytic streptococcus	2
12. Hemolytic clostridia	1
13. Coliform bacillus	1
14. Gram positive rod (in chains)	1

During the experiments on patients' skin, duplicate series of segments were obtained in some of the patients treated with tincture of acrizane, tincture of iodine, *p*HISOHEX, and zephiran. Inhibitors of the antiseptic agents used were added to the culture media in which the duplicate segments were cultured. Tamol, a substituted napthalene sulfonic acid was used as an inhibitor of acrizane; sodium thiosulfate as an inhibitor of iodine, and 2 per cent plasma as inhibitor of *p*HISOHEX and zephiran. Although there were more positive cultures in the initial culture tubes which contained the inhibitors, subcultures of tubes without inhibitors showed an equal number of positive cultures.

Since the efficiency of the preparation was calculated only on the basis of total inhibition of bacterial growth in these human studies, it was felt that the routine use of inhibitors was not necessary.

The efficiency of each skin preparation was indicated by the number of sterile segments found in each skin strip and not according to the degree of bacterial growth as used in the dog experiments. The average values for each group of patients, expressed as percentage efficiency, are shown in Table VI. The two antiseptic agents which demonstrated the greatest degerming activity were tincture of acrizane and tincture of iodine with efficiencies respectively of 77 per cent and 68 per cent. All other agents produced a significant reduction of the skin bacterial flora though to a lesser degree.

TABLE VI.—HUMAN STUDIES—THE EFFICIENCIES OF THE SKIN PREPARATIONS STUDIED

Antiseptic agent	Per cent negative cultures
Tincture of acrizane 1:200	77
Tincture of iodine 2%	68
Ether and chrysoidin Y solution	64
"5 cup preparation"	62
*p*Hisoderm hexachlorophene 3%	46.5
Tincture of merthiolate 1:1000	46.5
Chrysoidin Y alcohol solution	45.5
Aqueous zephiran 1:1000	36.5

The only disadvantage noted to the use of tincture of acrizane was the permanent staining of the drapes used at the time of operation. There were no toxic manifestations seen when tincture of acrizane was used as the agent for skin preparation.

On the other hand tincture of iodine did produce skin irritation over the prepared area on 4 of the 25 patients with production of vesicles on the skin of one patient. These toxic skin reactions occurred even though the tincture of iodine had been removed with 70 per cent ethyl alcohol prior to obtaining the skin specimens. The other antiseptic agents were not removed from the skin.

CONCLUSIONS

1. The efficiency of some commonly used methods of skin preparation is compared in a series of laboratory and clinical experiments.
2. An in vitro test indicates the antibacterial activity of the antiseptic agents against some of the bacteria commonly encountered on the skin.
3. Chick embryo studies compare the antibacterial activity of the antiseptic agents used with their tissue toxicity.
4. Animal studies show the effectiveness of the antiseptic preparations against a known and varied bacterial flora.
5. An in vivo method is presented which enables one to determine the effectiveness of an antiseptic agent in reducing both the transient and resident flora of the skin.
6. Tincture of acrizane 1:200 was found to be the most effective of the agents tested when used for preoperative preparation of the skin.

It also yielded the highest ratio of antibacterial activity to tissue toxicity, and was highly bacteriostatic in the in vitro tests.

NOTE.—Since the preceding studies were submitted for publication, tincture of acrizane, 1:200, has been used routinely in the general surgical operating rooms of the hospital of the University of Pennsylvania for a period of 1 month. Staining of the operating room linens has not proved to be a serious or objectionable disadvantage. In new unwashed drapes and towels, stains were produced which persisted after a single laundering but which disappeared on subsequent launderings. No stains were produced in the linens which had previously been laundered.

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ABERRANT SYSTEMIC ARTERIES TO THE LOWER LUNG

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ABERRANT systemic arteries to the lower lobe of the lung have been described occasionally as autopsy findings, and a total of 8 such cases have been collected (4) since the original report of Huber in 1777. The anomaly remained of only passing interest until the advent of pulmonary surgery, when the presence of a sizable artery in an unexpected location presented an added surgical hazard. The first surgical encounter with one of these vessels was reported by Harris in 1940, and in 1942 Haight warned that an anomalous artery must be suspected in any operation for suppurative or cystic disease of the lower lobe.

We have encountered at thoracotomy an aberrant artery to the lung in 5 patients; in 3 instances this vessel was associated with cystic abnormality of the lung, and in 2 cases with diaphragmatic hernia. These 5 cases have occurred in a series of 460 explorations of the chest for an incidence of 1.1 per cent. We are indebted to Dr. S. G. Robbins for the sixth case presented in this paper.

In view of the surgical significance of these vessels, the relatively small number recorded in the literature, and the interesting embryology of the defect, we wish to report our cases and summarize the embryology as it applies to the formation of the aberrant vessels and the associated anomalies.

CASE REPORTS

CASE 1. I. L. W., a white female, aged 20 years, was admitted to the West Tennessee Tuberculosis Hospital on October 11, 1950. She stated that her pulmonary symptoms started 8 years before when cough, sputum, fever, and hemoptysis followed an attack of measles. After x-ray studies, a diagnosis of lung abscess was made and an operation advised. The advice was not followed, and the patient recovered. She denied any symptoms during the 8 years between this first episode and the present illness, which started 3 weeks prior to admission. There was a sudden onset of cough, fever, and purulent sputum. These symptoms subsided after 3 days' treatment with sulfonamides, penicillin, and streptomycin, but hemoptysis then began and recurred repeatedly. Her condition slowly improved on continued bed rest, but after 3 weeks roentgenogram showed a density remaining in the chest (Fig. 1a). All studies for specific organisms were negative.

On admission examination, dullness and decreased breath sounds were present in the right base and x-ray examination confirmed the lesion confined to the distribution of the right lower lobe with some pleural reaction. The patient was afebrile. Bronchoscopy revealed normal major bronchi with bloody secretions from the right lower lobe orifices.

On postural drainage the chest lesion rapidly improved, but a large residual density remained. A tentative diagnosis of chronic suppurative disease of the right lower lobe, possibly complicated by congenital cystic abnormality was made and exploratory thoracotomy performed on November 6, 1950. The disease process was found to be limited to the lower lobe. The pulmonary arterial branches were grossly normal. Below and posterior to the inferior pulmonary vein, a 1 centimeter artery entered the lower lobe. It arose from the aorta and crossed behind the esophagus. It was doubly ligated and divided, after which the lobectomy was completed in the usual fashion. The postoperative course was uneventful, and the patient was dismissed on the twenty-first postoperative day.

The surgical specimen showed a cyst measuring 6 centimeters with several bronchial communications (Fig. 2). The anomalous artery was injected and its branches arched about the cyst and anastomosed freely with the pulmonary arterial capillary branches.

CASE 2. M. R. B., a 43 year old white female, was admitted on March 16, 1949. Her health was good. In the course of a routine x-ray survey of all school teachers, a shadow was seen in the right chest, and the patient was sent in for further investigation. There was a history of one episode of mild pleurisy in the right side in 1935, 14 years prior to the discovery of the lesion in the lung. Chest x-ray film in 1935 was said to be normal, but it is unavailable for comparison. Physical findings were all normal. On x-ray examination, a rounded mass measuring 5 by 7 centimeter was seen opposite the ninth rib posteriorly (Fig. 1b). Exploratory thoracotomy was done on March 21, 1949. A cystic tumor mass was palpated within the substance of the lower lobe. The remainder of the lung was normal. An anomalous artery 1.5 centimeters in diameter was discovered

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arising from the aorta just above the diaphragm coursing upward and anteriorly to enter the diaphragmatic surface of the lobe. The pulmonary artery appeared somewhat smaller than usual. There was a branch from the inferior pulmonary vein to the middle lobe. The anomalous artery was doubly ligated and divided, and the lobe then removed in the usual manner.

Postoperative course was uneventful and the patient was dismissed from the hospital on the sixteenth postoperative day.

On examination of the surgical specimen, a 6 centimeter cyst was found in the lobe with no bronchial communication. It was lined with bronchial epithelium and filled with a gelatinous mucoid material. The adjacent pulmonary tissue showed chronic pneumonitis. The anomalous artery was injected and appeared to supply the cystic area of the lobe. No pulmonary arterial supply to this region could be demonstrated.

Case 3. E. E. O'F., a white female, 23 years old, was admitted to the Methodist Hospital on February 13, 1949. She stated that her health had always been good, and she led quite an active life, playing basketball up to the time of her admission. Three years before her hospitalization, she was involved in an automobile accident and had received superficial lacerations and considerable "shaking up." She recovered promptly from this episode but had since noticed occasional bouts of dyspnea and slight chest pain. A routine chest survey x-ray examination in December, 1948, showed an abnormal density in the right chest, and she was referred in for examination.

She was found to have no abnormalities other than that revealed by the chest film: a dense, rounded mass just above the diaphragm in the right posterior chest (Fig. 1c). The diaphragm moved, and this mass appeared to move with it to a smaller degree. Exploratory thoracotomy on February 14, 1949 revealed a diaphragmatic hernia of the posterior type, apparently traumatic in origin. The liver was partially herniated through, as was the kidney. From the region of the kidney, an artery 5 millimeters in diameter ran up through a fold of pleura into the lower lobe. This was a systemic vessel. The pulmonary artery was dissected in the fissure enough to demonstrate its normal size and distribution, after which the anomalous artery was divided. The defect in the diaphragm was then repaired without incident, and the patient convalesced nicely. She has been without symptoms since the operation.

Case 4. Rev. R. R. D., a 50 year old white male, was in good health until 6 months before admission at which time a severe attack of influenza occurred and left the patient weak. He had since lost 20 pounds and had had recurrent attacks of painless hoarseness. There were no other symptoms and no history of injury at any time. Physical examination was essentially normal. On x-ray examination of the chest, a rounded density 10 centimeters in diameter was seen in the cardiohepatic angle (Fig. 1d). This mass appeared inseparable from the diaphragm, and moved with the diaphragm on respiration, though to a lesser degree. It was thought to be a localized eventration of the diaphragm. A pneumoperitoneum was introduced, and further x-ray studies revealed that there was a localized area of eventration with molding of the surface of the liver. On June 19, 1940 exploratory thoracotomy was done, and the eventration confirmed. A portion of the liver and the right kidney were in the eventrated portion of the diaphragm. There was a pulsating, thick walled vessel piercing the diaphragm just posterior and medial to the area of eventration and entering the lower lobe below and behind the inferior pulmonary vein. The oblique fissure was developed sufficiently to map out an entirely normal pulmonary arterial distribution. The pulmonary tissue was carefully examined, and no abnormalities found. The anomalous artery was then doubly ligated and divided, and the eventration of the diaphragm repaired.

Postoperative course was uneventful, and the patient was discharged on the twelfth postoperative day. He has remained without symptoms since his operation, although no reason for the hoarseness and weight loss was discovered.

Case 5. J. H. D., a white male, 28 years old, was admitted February 13, 1950, with a history of cough, purulent sputum, and frequent episodes of pneumonia dating back to a severe case of measles at the age of 5. There is also the history of idiopathic epilepsy under treatment with dilantin and phenobarbital. On examination, there were rales in the left base posteriorly, and bronchograms revealed bronchiectatic changes limited to the left lower lobe (Fig. 1 e, f). At bronchoscopy, purulent secretions were seen to issue from the lower lobe on the left side, while all other orifices appeared normal. At the time of exploration of the chest, tremendous quantities of purulent secretions were aspirated after tracheal intubation. The chest was entered through the bed of the seventh rib, and the lower lobe was found to be adherent throughout. Arising from the aorta and entering the posteromedial aspect of the lobe was an aberrant artery 5 millimeters in diameter. This was doubly ligated and divided after which the lobe was removed in the routine manner. The pulmonary circulation to this lobe was normal.

Convalescence was uneventful, and the patient was discharged to his home on the ninth postoperative day. Pathological examination disclosed the aberrant artery in intimate relation with a 9 by 5 by 4 centimeter cyst in the substance of the lobe. The adjacent bronchi showed cylindrical and saccular bronchiectasis.

Case 6. A. D., a colored male, aged 3 years, was admitted on March 26, 1951 with a history of high fever for 2 days, cough and chest pain for 1 day. He had had measles the preceding month. Upon admission to the hospital his temperature was 105 degrees F. and the chest roentgenograms showed a pneumonic infiltration in the lower left lung field being most marked medially. After several days of chemotherapy the symptoms subsided but a density con-

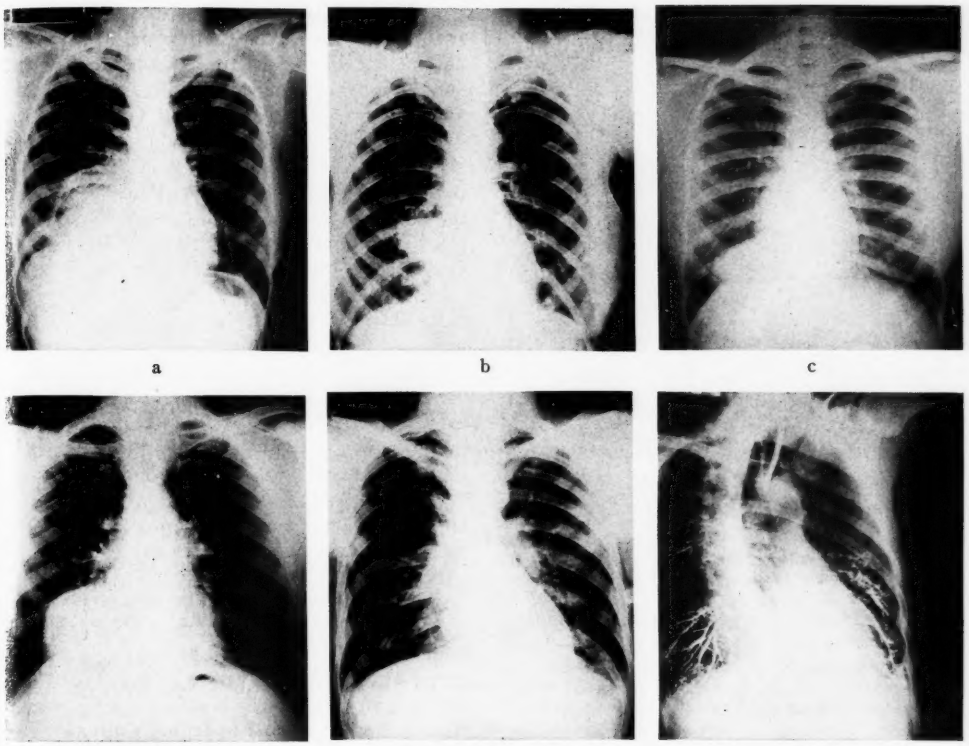


Fig. 1. a, Case 1. Preoperative chest x-ray film showing cystic density remaining after a pneumonic episode. At operation, the cystic structure resembled a marked segmental bronchiectasis filled with old blood. b, Case 2. Preoperative chest film shows a rounded density in the right lower lung field. The cyst was filled with mucoid material and was without bronchial communication. c, Case 3. Chest film showing herniation of right diaphragm. Kidney and liver were through the hernia and an aberrant artery coursed through the defect in the diaphragm to a normal lung. d, Case 4. Chest film showing larger density in right base, proved by pneumoperitoneum to be eventration of diaphragm. At operation, an aberrant systemic artery ran through the diaphragm to a normal lower lobe. e, f, Case 5. Preoperative chest film and oblique view of bronchogram. Bronchiectatic changes are visible, but the cyst cannot be seen behind the heart. The cyst communicated freely with the bronchial tree.

taining cystic areas remained in the posterior basilar portion of the left lower lobe (Fig. 3a). Bronchoscopic examination disclosed secretions from the left lower bronchus and an inflammatory reaction in the left main bronchus opposite the upper lobe bronchial orifice. Bronchograms were unsatisfactory. A left lower lobectomy was performed on May 24, 1951. An aberrant artery originating from the lower thoracic aortic and passing to the medioposterior base of the lower lobe was divided. The postoperative course was uneventful.

After surgical removal the lower lobe could be readily inflated through the segmental bronchi except for part of the cystic area in the medioposterior basilar portion. When the specimen was injected by the intrabronchial introduction of formalin under low gravity pressure this same area failed to fill but after submerging the specimen a few hours in a container of formalin the cystic zone became distended. In the unfixed air-inflated specimen diodrast introduced into the anomalous artery flowed freely from the pulmonary vein. Roentgenograms of the specimen show the diodrast-filled vessels arching about the cystic areas. A more viscid radio-opaque medium consisting of lead chromate suspended in a solution of methyl cellulose revealed the branches of the anomalous artery passed to all areas of the lobe, including the unaltered portions (Fig. 3c). The colored solution injected into the aberrant artery oozed from the pulmonary artery in the upper part of the lobe but not from a pulmonary artery passing to the cystic area. The three surgically divided bronchi at

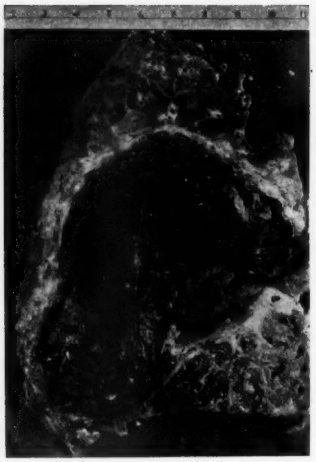


Fig. 2. Case 1. The large cystic structure filled with old blood is actually an area of segmental bronchiectasis with narrowed proximal bronchi. The group of smaller cystic structures in the lower right represents a separate area of segmental bronchiectasis. The aberrant artery from the aorta passed to both of these segments.

the hilum had no demonstrable communication with the cysts. These cysts actually resembled dilated portions of a segment of the bronchial tree with less dilated ostia representing the intercommunications. Considerable mucus filled most of the cysts.

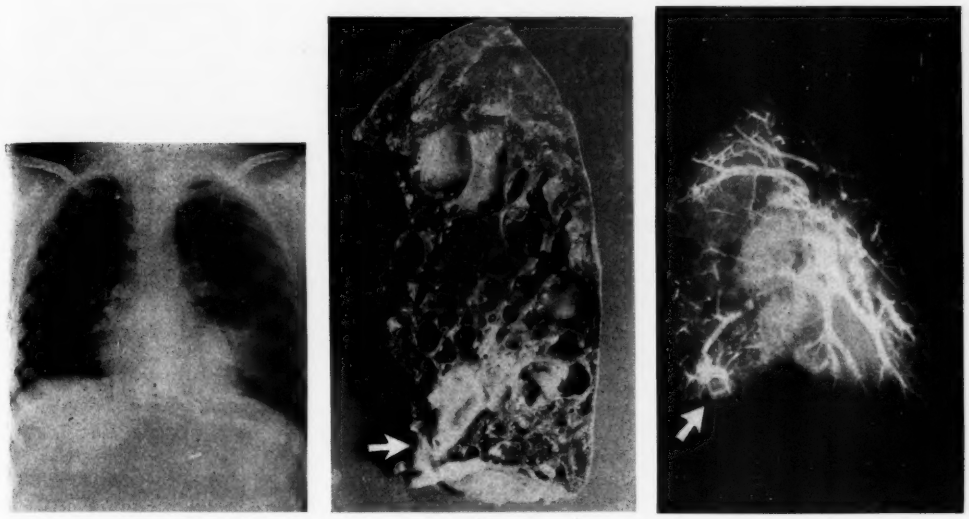
Aberrant arteries to the lung have repeatedly been described as piercing the diaphragm, and in a recent report by Clay and Hanlon one such vessel is described in association with diaphragmatic eventration. Clay and Hanlon elected to preserve the artery, repairing the diaphragm up to the point of entry of the vessel. In both our cases of diaphragmatic defects the pulmonary circulation was investigated sufficiently to establish its normal size and distribution, and the anomalous artery then was sacrificed. This procedure facilitates more complete repair of the diaphragm. In addition the recirculation of arterialized blood through the lung is, in effect, an arteriovenous shunt and serves no useful purpose. No untoward reactions have followed division of the artery.

Including the 6 cases presented we know of 57 cases of anomalous arteries to the lower lung found at operation, 42 of these now having been reported in the literature. Bruwer, Clagett, and McDonald in 1950 reviewed 30 cases, including 5 of their own. Two more cases are reported in the case records of the Massachusetts General Hospital, 1 by Finley and Maier, 2 by Tosatti and Gravel, and 1 mentioned by Clay and Hanlon in their recent article on diaphragmatic hernia. In addition to these 36 reported cases, Bruwer and his associates mention 9 unpublished cases as known to them. Carr and Skinner have 1 case and Hughes has 5, all encountered during surgical exploration of the chest.

In all of the 42 cases of which clinical summaries are known to us, the anomalous artery per se has caused no disability, but an associated abnormality has been sufficient to indicate operation. In 45 cases, pulmonary findings were present, and in 3 there was a defect in the diaphragm. For an intelligent discussion of the development and association of these anomalies, a review of the embryology of the lung and lower chest is necessary.

EMBRYOLOGY

The lung bud first appears as a ventral outpouching from the foregut in the 3 to 3.5 millimeter human embryo (18 to 21 days) (1). The embryo at this time is a soft, delicate, transparent mass of tissue in which dramatic and rapid changes are occurring in structural relationship and in size. The major vessels are little larger than capillaries, and the future thoracic area consists of growing mesenchymal cells interlaced with an abundant vascular plexus. The foregut passes through the posterior part of this mesenchymal mass and is intimately surrounded by the capillary splanchnic plexus which has numerous connections with the dorsal aorta. The lung and liver buds arise in close proximity from the ventral surface of the foregut. They lie behind the heart and project into the mesenchymal mass, carrying with them a network from the splanchnic plexus. By the time the embryo has reached 8 millimeters, the caudal end of the lung bud has separated from the foregut and has divided into primary bronchi, with 2 lobe buds on the left and 3 on the right (21). The mesenchymal interstitial tissue into which these buds project has responded by localized overgrowth about the tips of the de-



a b c

Fig. 3. a, Case 6. After relief of pneumonia in a 3 year old child a residual density remained in the lower lobe. b, Most of the cystic, dilated bronchi were filled with mucopurulent material which has been removed revealing the smooth walls and narrow intercommunications. The aberrant artery enters the base of the lower lobe in its mediposterior aspect (see arrow, b and c). c, Roentgenogram of artery injected with lead chromate suspended in viscid methyl cellulose solution shows the extensive distribution of this vessel.

veloping lung bud, which eventuates into the division of the lung into lobes. The vascular plexus in the primitive lung loses its communications with the splanchnic plexus and the dorsal aorta during the caudal and ventral growth of the lung and the formation of the pleural cavities. The only dorsal arteries which normally persist in the lung are the bronchial arteries. The embryologic development of these variable bronchial arteries has not been described in any detail. The pulmonary arteries and veins apparently develop from the capillary plexus in the primitive lung before any attachment to the incomplete aortic arch system exists. This has been noted by Buell in the chick, Huntington in the cat, Bremer in a variety of mammals and Congdon in the human embryo.

Meanwhile the liver bud rapidly enlarges, growing ventrally from the foregut into a mesenchymal mass below the heart. This mesenchymal tissue splits, the lower part becoming the stroma of the liver and the upper the septum transversum, which is the earliest and most anterior part of the diaphragm. The ventral growth of the liver and the interposition of the primitive diaphragm in all probability contribute to the interruption of the vascular connections between the mesenchymal tissue of the primitive lung and that of the liver.

The pleural, pericardial, and peritoneal spaces have formed by splitting of the mesoderm, and the pericardial space has been enclosed. The pleural and peritoneal spaces remain in communication dorsally through the pleuroperitoneal iter. This communication is not closed until the embryo reaches 18 to 19 millimeters (21); it is the last of the diaphragm to be formed and is the site of congenital diaphragmatic defects.

In summary, the basic framework of the lungs, pleural space, and diaphragm are developed between the 3.5 and the 19 millimeter stage in embryo. This period of growth covers 30 to 32 days, but increase in bronchial subdivisions continues. At birth there are 17 generations of bronchi, and by middle childhood the maximum division of 24-24 bronchial branchings is attained.

DISCUSSION

Anomalous systemic arteries to the lung have originated from the aorta and all of its major branches, both thoracic and upper abdominal. This subject has been comprehensively reviewed by Finley and Maier. They found 67 cases in which sufficient information was available to permit analysis. Of these 25 (37.3 per cent) arose from systemic arteries above the lung roots, while the remaining 42 (62.7 per cent) arose below the hilar level. This division with reference to the pulmonary hilum is of value as there are differences in the embryologic development and the clinical implications in the two groups. The suprahilar arteries may possibly be compensatory collateral vessels (from decreased pulmonary arterial flow) since 75 per cent were associated with other major anomalies of the heart and great vessels; none were associated with anomalies of the lung. On the other hand, only 16 per cent of the infrahilar aberrant arteries were found to accompany other cardiovascular anomalies while in 70 per cent anomalies of the lung were present.

It follows, then, that the surgical interest in the two groups of anomalous arteries differs. Those arteries arising above the pulmonary hilum will be encountered during operations for correction of cardiovascular anomalies, while the infrahilar vessels will be found during operation for pulmonary and diaphragmatic lesions. It is with these infrahilar aberrant arteries and the associated anomalies of the lung and diaphragm that this paper is concerned.

The theories that have been proposed to account for the anomalies involving the lower lobe, its blood supply, and the diaphragm are comparable to those given by Davis and Gunz for accessory lung, with the addition of the recent theory advanced by Pryce (23, 24).

1. Vascular
   a. Pressure of the embryonic aortic arch system producing deficiency of bronchial system (Vogel)
   b. Constriction of portion of normally developing lower lobe analogous to formation of azygos lobe (Humphreys)
   c. Anomalous artery "captures" a portion of lung bud causing it to sequestrate (Pryce, 23)
2. Bronchi or primitive pulmonary tissue
   a. Reduplication; development of accessory bud from digestive tract (Eppinger)
   b. Deepening of normal or abnormal pulmonary tissue (Retoryk)
3. Diaphragm; amputation of pleuroperitoneal iter
4. Embryonic adhesions between lung bud and adjoining tissue (Cockayne and Gladstone)
5. Teratomatous inclusion

In general these theories attribute the entire group of anomalies to the influences of one morphologic structure, and differ only in whether the primary or determining role of the bronchi, the blood vessels, or the diaphragm is emphasized.

The most recent theory, proposed by Pryce (23) in 1946, ascribes the primary role to the aberrant artery which "captures" one or more bulbous tips of the developing lung bud. Subsequently this leads to sequestration (dissociation) or death of the "captured" primitive pulmonary tissue, thereby giving rise to congenital cysts, lower accessory lung, and pulmonary agenesis depending on the size of the lung bud which is captured. He also proposed that ischemic traction of the abnormal embryonic arteries from the developing diaphragm to the lung might give rise to congenital defects of the diaphragm. This theory is attractive in that it ties together this widely varying group of anomalies. However, it runs counter to the concepts of Beneke that a displaced organ acquires a blood supply from the adjoining embryonic tissue and to the principles of vascular growth as formulated by Thomas in 1893 and by Evans in 1909. In general the various theories relate the entire anomaly to the influence of one structural abnormality without attempting to explain the latter itself. Current knowledge of the principles of embryologic growth is not adequate to explain any anomaly.

The pattern of embryonic differentiation results from an integration of poorly understood organizing influences which, though complex, seldom fail to repeat the species pattern. Any "imbalance of influences" in a certain area could result in anomalies of all morphologic structures under development in that area at that time. Such defects have been ascribed to infection, altered nutrition, or genetic factors, mediated through chemical as well as morphological means. Needham presents the concepts of biochemical morphogenesis. For example, genetic influences act

through inductors which do not diffuse through cell membranes but may alter the cellular production of hormones, catalysts, or inhibitors of diffusible type. Apparently chemical mediators have not been described in the region of the lung.

It would appear that one cannot ascribe any primary role to either the pulmonary tissue, the blood vessels, or the diaphragm. The most likely explanation is that some interference with the embryologic organization takes place in the lower chest behind the heart and that all structures under development in that area at that time may be affected. This would include the lower pulmonary tissue, especially the lower lobe, the diaphragm in its posterior portion at the region of the pleuroperitoneal fold, and remaining communicating small vessels between the old pulmonary plexus in the dorsal aortic zone.

In consideration of the varying association of anomalies involving congenital cyst of the lung, infrahilar aberrant systemic artery to the cystic area, and congenital diaphragmatic defects, the following points appear important:

1. Congenital bronchogenic cysts of the lung may be found anywhere in the lung. They may develop at any time during the period of active bronchial subdivision, which lasts through middle childhood. Only a few of those cysts in the lower lobe are associated with the abnormal artery.

2. Suprahilar aberrant systemic arteries to the lung have not as yet been demonstrated in association with congenital bronchogenic cysts.

3. An aberrant artery may be associated with diaphragmatic defects without any accompanying anomaly in the pulmonary tissue. The diaphragmatic defect is in the region of the pleuroperitoneal membrane.

4. The infrahilar aberrant arteries enter the lung in the posteromedial aspect of the lower lobe which is adjacent to the pleuroperitoneal membrane.

5. Infrahilar aberrant systemic artery has apparently not been reported as an isolated anomaly. The other defects in this group may occur separately or in all combinations together.

SUMMARY

1. Six cases of aberrant systemic arteries to the lower lobe of the lung are reported. We have knowledge of 57 such cases discovered at operation, 42 of them having now been recorded in the literature. This anomaly is much more common than indicated by the earlier literature.

2. The aberrant systemic arteries originating from the lower thoracic or upper abdominal aorta may be associated with accessory pulmonary lobes, cystic disease of the lower lobe, and diaphragmatic defects. In 2 of the 6 cases reported here diaphragmatic defects were present while in the remaining 4 cases there was cystic disease of the lower lobe. The clinical findings depend upon the presence or absence of bronchial communication to the cystic area. With bronchial communication infection occurs and the findings of marked segmental bronchiectasis are present. In our 1 case without bronchial communication no symptoms were present.

3. Only those aberrant vessels arising below the level of the pulmonary hilum are associated with the cystic disease and diaphragmatic defects. Those originating above the pulmonary hilum are present in anomalies of the heart and great vessels.

4. The embryologic development of the lower thoracic area is reviewed and the theories pertaining to the formation of associated anomalies are discussed. These theories stress the determining role of one morphologic aspect without attempting to explain the original developmental error. Present knowledge is inadequate to explain any anomaly but the association of anomalies of the lung, systemic vessels, and diaphragm suggests a common developmental error involving all structures in the lower thoracic area.

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RECTOCOLIC ANASTOMOSIS

A Simplified Method

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THAT resection of the rectum with rectocolic anastomosis occupies a definite place in colonic surgery, few would any longer contest though any discussion as to its contraindications and scope would undoubtedly arouse a diversity of opinion. Our own observation and experience have convinced us that rectocolic anastomoses are frequently done too high in the rectum and frequently too close to the tumor because of the awkwardness of suturing two ends of a contaminated structure low down on the pelvic floor. Especially is this true in small male pelves. That the difficulties imposed by this combination of an adverse anatomical area and infective structures have been shared by others seems to be well affirmed by the variety of methods, old and new, which have been revived and devised to better control the increased morbidity and the occasional additional mortality which has resulted from these attempts to save the anal sphincter.

It is our intention to present here still another method of anastomosis. We believe that it may quite properly be recommended for its greater simplicity, for the facility with which the anastomosis may be accomplished, for the negligible amount of contamination involved, and for the security which is afforded against separation of the bowel ends in the postoperative period. The method depends on the use of a special clamp which, when properly assembled, establishes and maintains the anastomosis, crushes its way free from the engaged tissue at a suitably chosen time in the postoperative period, and meanwhile provides for the escape of gas and stool. Finally it may easily be withdrawn through the anus.

In general, the instrument consists of three parts (Fig. 1). Part A inserts into the end of the proximal colon. Its salient features are its shape with an indentation at the midportion of its body to permit easy handling and to provide better purchase while it is contained within the bowel; spaced needles on which the bowel end is impaled; holes for interdigitation with the needles of part C; a lumen with threads at its proximal end for insertion and engagement of part B and a small key to assure accurate engagement of all needles with their respective holes in parts A and C.

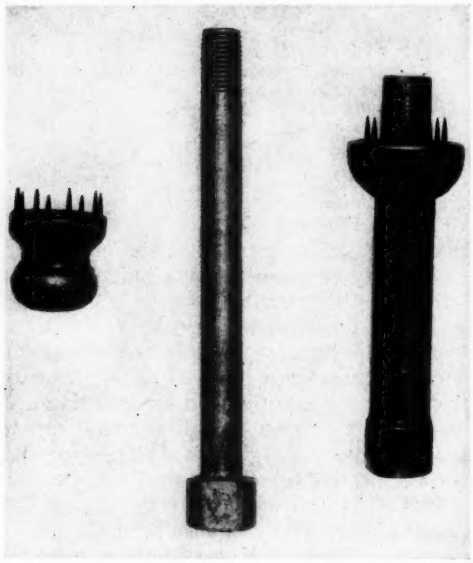
Part C inserts into the rectal stump. Its proximal portion is similar in most respects to part A with which it meshes to appose the bowel ends. A distal flanged hollow stem passes through the anal sphincter from within outward and is grasped beneath the drape just outside the anus.

Part B is a tubular structure with threads at its upper end for engagement with the threads of part A. A flange at its lower end is provided for impingement on a similar flange at the lower end of part B. It is this flange which first maintains parts A and C in close apposition and which, later as the clamp is tightened, provides it with the ability to crush through the tissue contained between them. The lumen provides for the escape of gas, mucus, and stool.

The clamp has been used with great satisfaction in a number of patients. Several case reports indicating to some extent the variety of its uses will be presented.

CASE 1. C.M., an 80 year old physician, was first seen on May 10, 1949. He complained of pain in the lower abdomen, poor appetite, and increasing constipation with bloating for one year. He had lost 15 pounds in weight over a 6 month period. Examination was not remarkable except for a palpably hard, nodular, annular mass just at the tip of the examining finger. A chest x-ray was reported as negative for metastases.

He was prepared for operation with two blood transfusions and sulfasuxidine was also given. On



A B C

Fig. 1. Photograph of parts of the anastomotic instrument (slightly smaller than actual size).

May 21, 1949, exploration showed no evidence of distant abdominal metastases with the tumor approximately 4 centimeters above the cul-de-sac reflection. The bowel proximal to the tumor was hypertrophied and contained more than the usual amount of stool. The lower half of the sigmoid and the upper two-thirds of the rectum were resected with the entire inferior mesenteric vascular stalk. A drain was brought out through the right ischiorectal fossa, its tip lying in the presacral space. Anastomosis was quickly accomplished over the rectal clamp. Because of the partial obstruction, it was felt advisable to perform a proximal transverse loop colostomy.

Pathological study of the bowel showed a segment of bowel measuring 24 centimeters in length.[1] The mesenteric stalk measured 13 centimeters from the upper border of the tumor to its cut end. When the bowel was opened an annular carcinoma was noted, the lower border being 7.5 centimeters from the distal end of the specimen. Histologically the tumor was a moderately well differentiated adenocarcinoma. Sections of the lymph nodes were negative for evidence of metastases.

Postoperatively the patient did well, the rectal clamp was tightened on the third and sixth postoperative days and was removed on the ninth day. He was readmitted 2 months later and the loop colostomy was closed.

[1]Measurements made in the laboratory on tissues which had undergone contraction.

He has been seen at frequent intervals since operation. For a few months he had had a feeling of urgency when desire to defecate occurred. This feeling has subsided. The anastomosis admits the examining finger easily and a barium enema shows an adequate lumen with no retention of barium on evacuation. There is no stricturing at the anastomotic site. When last seen on March 28, 1951, there was no evidence of disease.

This case represents an ideal situation: an aged patient in whom the increased strain of a Miles resection is to be avoided and a lesion safely located for anastomosis if this can be done low down on the rectum with minimum contamination.

CASE 2. H.S., a 61 year old white female, was admitted to hospital on September 7, 1950, complaining of bloating and "pencil-like" stools for 1 month. Physical examination was not remarkable. A stool specimen examined for blood showed a four plus guaiac reaction. Barium enema showed an annular filling defect in the midsigmoid area. Patient was given sulfasuxidine and streptomycin postoperatively. On September 12, 1950, exploration revealed no distant metastases. The inferior mesenteric vessels were divided just beyond the point of emergence of the artery from the aorta. An anterior rectosigmoid resection was performed with anastomosis approximately 2.5 centimeters above the levator ani muscles, the anastomotic clamp being used. A drain was placed in the presacral space and brought out through the posterior fornix of the vagina. No obstruction of the bowel had been present and the bowel was well cleansed. Accordingly, it was not felt necessary to do a proximal temporary colostomy.

The resected bowel segment measured 28 centimeters in length. The attached rectal vascular stalk or mesentery measured 12 centimeters in length. When the bowel was opened an annular lesion was found, the lower edge of the tumor being 10 centimeters from the distal end of the rectum, the upper border of the tumor 14 centimeters from the proximal end of the resected bowel. Microscopically the tumor was an adenocarcinoma, grade II, and the regional nodes were negative for metastatic tumor.

The postoperative course was uneventful. The patient was discharged on the fourteenth postoperative day with the vaginal drain still in place. The drain was removed approximately 6 weeks postoperatively. Digital examination of the rectum shows a very adequate lumen at the site of anastomosis. On April 2, 1951, when last seen there was no clinical evidence of persistent disease.

This was an ideal situation for the performance of a conventional V-type of sigmoid removal in which the inferior mesenteric vessels are of necessity spared. In this instance, however, the inferior mesenteric as well as the sigmoidal node chain was removed.

Fig. 2. Incision of the lateral leaf of the sigmoid mesocolon is carried out and identification is made of the left ureter.

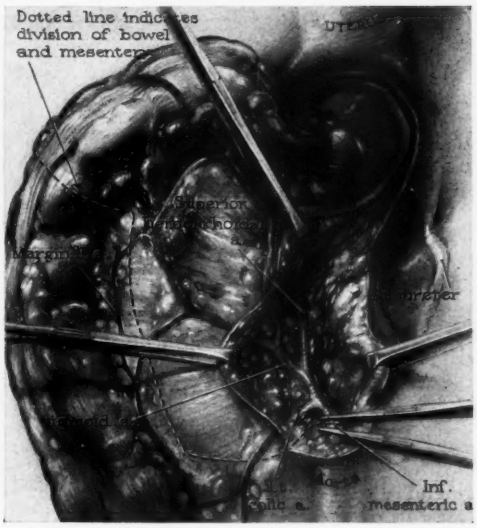


Fig. 3. Incision of medial leaf of sigmoid mesocolon; clamping stump of inferior mesenteric artery; and proposed line of incision of the colonic mesentery.

CASE 3. L.P., a 68 year old white male, was first seen by us on December 18, 1949 in consultation. The patient had previously had a transverse colostomy for intestinal obstruction. The surgeon stated that he had palpated a hard mass in the lower sigmoid area. No rectal lesion could be palpated but barium enema showed a large filling defect in the sigmoid area.

On January 5, 1950, he was explored. In the lower sigmoid a large hard mass was found densely adherent to the dome of the bladder and to the left pelvic wall and was considered to be typical of a carcinoma of the low sigmoid invading the bladder fundus. No distant metastases were found. The proximal sigmoid was extremely thickened and edematous. With considerable difficulty the mass was freed and resected with the bladder fundus approximately 7 centimeters above the levator ani muscles. Because of the hypertrophy and edema of the cut end of the proximal sigmoid colon, it was not felt suitable for any type of anastomosis. The rectal stump was closed and the sigmoid brought out through the right rectus muscle splitting incision as an end colostomy.

Examination of the resected specimen showed a 20 centimeter segment of bowel with a firm mass in the center of the specimen. The overlying serosa was puckered and red in color. The mass was very hard to palpation. The bowel was opened, and marked stenosis was noted at the site of the mass, with almost complete occlusion of the lumen but the mucosa was intact. The bowel wall was thickened and cut almost like cartilage. Several small abscess cavities were noted in the thickened bowel wall. The proximal bowel was edematous and greatly dilated. Microscopic study showed extensive inflammatory changes with considerable hyalinized fibrous tissue. The lymph nodes showed only hyperplasia. The diagnosis was interstitial colitis with extensive fibrosis.

Postoperatively the patient did quite well and he was discharged from the hospital on the nineteenth postoperative day.

He was readmitted on March 12, 1950 and the following day the transverse colostomy was closed in order to provide better bowel control.

The patient was again admitted to the hospital on October 17, 1950 for re-establishment of rectal continuity. On the following day the sigmoid colostomy was torn down and the rectal stump was opened. Anastomosis between the sigmoid colon and rectal stump was accomplished over the rectal anastomotic clamp. Postoperative course was uneventful.

When last seen on February 8, 1951 the patient was having two to four bowel movements daily without difficulty. The anastomosis admitted the examining finger snugly.

CASE 4. H.H., a 42 year old white male, was first seen on July 26, 1948, complaining of passing blood per rectum for several months. He also complained of pain radiating down into the penis. Physical examination was not remarkable except for tenderness in the left lower quadrant of the abdomen. He was admitted to the hospital and a barium enema showed a filling defect in the lower sigmoid colon.

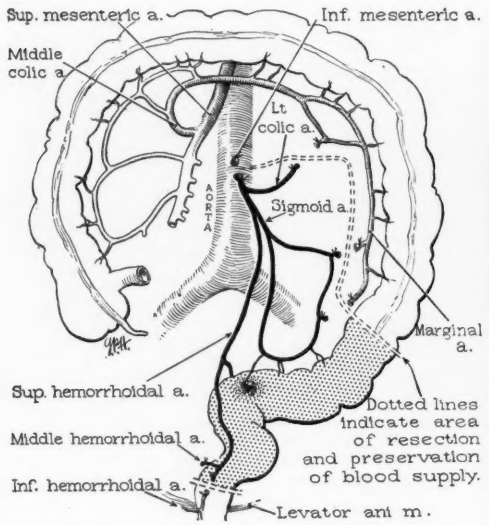


Fig. 4. Graphic representation of the area to be removed and usual manner of preservation of left colonic blood supply.

Fig. 5. Inferior mesenteric stump ligated and bowel mesentery divided; tape placed about bowel at site selected for anastomosis.

Proctoscopic examination showed what appeared to be a typical carcinoma 22 centimeters from the anus. The bowel was prepared with sulfasuxidine and on August 2, 1948, exploration was performed. There was no evidence of distant abdominal metastases.

Fig. 6. Development of anterior rectal plane.

A large tumor in the lower sigmoid was present and fixed to the entire posterior aspect of the bladder. It was obvious that if resection was to be performed the bladder would also have to be sacrificed. The lower two-thirds of the sigmoid and upper two-thirds of the rectum with the attached mesentery, bladder and prostate were resected. Anastomosis was then carried out over the rectal clamp, the sigmoid colon being under a little tension. The ureters were implanted in the sigmoid colon.

Pathological study of the specimen showed a segment of bowel measuring 21 centimeters in length with the attached bladder, prostate, and inferior mesenteric stalk. The bladder was opened and bullous edema of the bladder mucosa in the area of the trigone was noted. The bowel tumor was annular in character; its lower border was 55 centimeters from the cut end of the rectum. The mesenteric stalk measured 14 centimeters in length. Histologically the tumor was an adenocarcinoma, grade II. The bladder showed invasion of the muscularis. No tumor was found in the lymph nodes examined.

The rectal clamp was tightened on the third and sixth postoperative days. It became loose and was removed on the eighth postoperative day. Rectal examination a few days later showed separation of the posterior part of the bowel at the anastomotic site. A transverse colostomy was performed on September 9, 1948, to divert the fecal stream so that only urine would be present in the lower bowel. On October 6, 1948, the coccyx was removed and the posterior opening in the bowel was closed. The anastomosis then proved to be watertight.

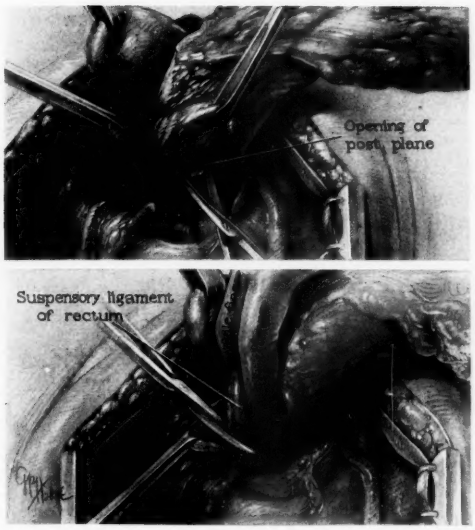


Fig. 7. Development of posterior rectal plane and division of suspensory (lateral) ligaments of rectum.

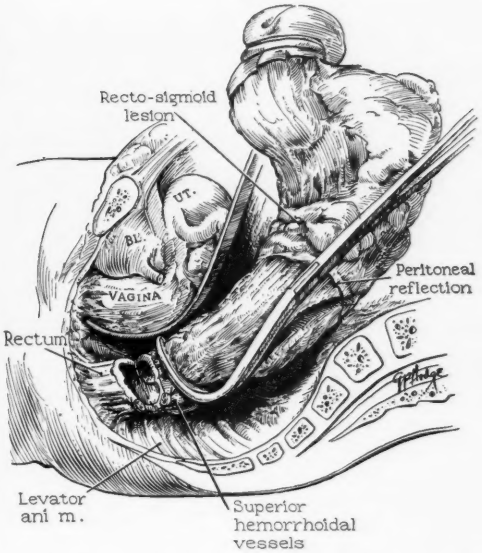


Fig. 8. Ligation of hemorrhoidal vessels above the levator muscles with beginning transection of the rectum.

The patient was allowed to go home but was readmitted on May 8, 1949, for colostomy closure.

He was seen at frequent intervals and his chief complaint was difficulty in holding urine and stool at night, having to get up about every 2 hours. On March 29, 1950, examination showed two small hard supraclavicular nodes and a small nodule in the epigastric area thought to be in the liver. These masses increased in size on subsequent visits and the supraclavicular nodes obviously represented metastatic disease. He expired on October 1, 1950, 20 months following extirpation of his tumor.

The usual alternative in this patient would have been a wet colostomy. This was the first patient in whom the clamp was used. Freeing up of the splenic flexure would have undoubtedly prevented separation at the anastomotic site.

DISCUSSION

We do not deplore a colostomy and readily agree that any attempt to save the anal sphincter may be abused. We unequivocally believe that any risk to the curability of a patient involved in saving the sphincter is wholly unjustified. In general our use of the clamp in colonic cancer has been limited to tumors above the cul-de-sac level. The markedly lessened strain imposed on the patient by this type of procedure as compared with a Miles resection impelled us on one occasion to violate this rule on a 79 year old female who had recently been treated for severe cardiac decompensation. We did not feel that this

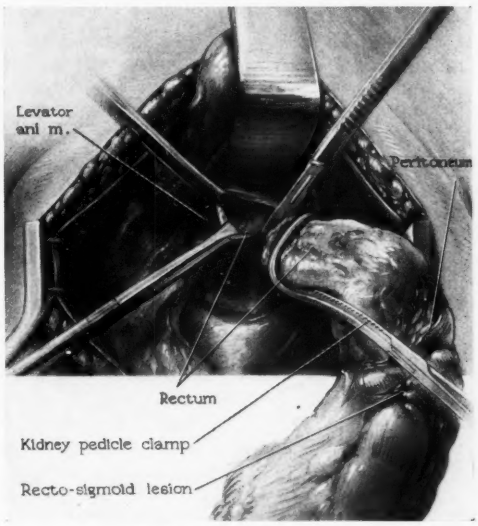


Fig. 9. Technique of lower rectal division.

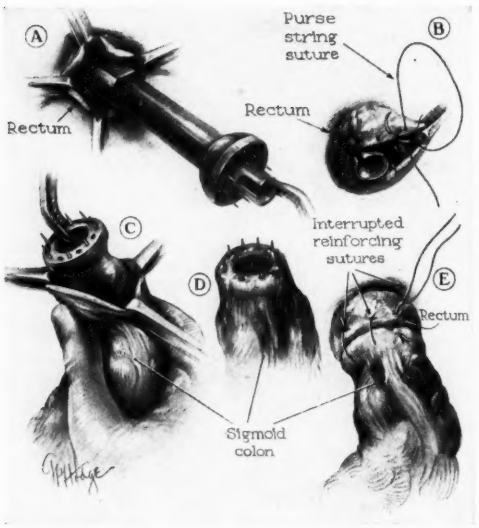


Fig. 10. A, Part C of instrument being inserted through rectal stump; B, pursestring suture to adapt greater circumference of rectum to that of the sigmoid; C, part A of instrument being inserted into open end of sigmoid; D, sigmoid impaled on needles; E, few interrupted sutures placed after parts C and A are held in apposition by screw-stem B.

patient could withstand a Miles type of resection. The tumor was bulky, involved the upper rectal ampulla, and was fixed to the pelvic wall. Microscopic examination disclosed that it had involved all layers of the rectal wall and had metastasized to numerous lymph nodes. Though the anastomosis was made at the levator level, a recurrence took place which seemed to derive from the bowel wall, though we could not be sure. At any rate, we strongly believe that any tumors of the upper rectal ampulla, unless confined to the mucosa and arising in a polyp, should be treated by Miles resection. We do not believe that any rectal cancer, even the earliest, lying below the upper ampulla should be treated by any form of sphincter saving attempt at removal. It has been our impression that failure to find tumor contained within lymphatics at certain distances from the primary site (1, 4) as has recently been reported, is small assurance against the possibility of recurrence when one considers the defections inherent in even the most careful histologic examination of tissue. There seems to be fairly uniform agreement (2, 3, 5) that retrograde lymph node involvement is infrequent and is apt to occur only when the lymphatic channels proximal to the tumor are plugged. Moreover, our own observations relative to bowel wall involvement, especially in the rectum, have led us to do resections of adjacent structures in increasing numbers whenever any fixation of tumor is present (6, 7).

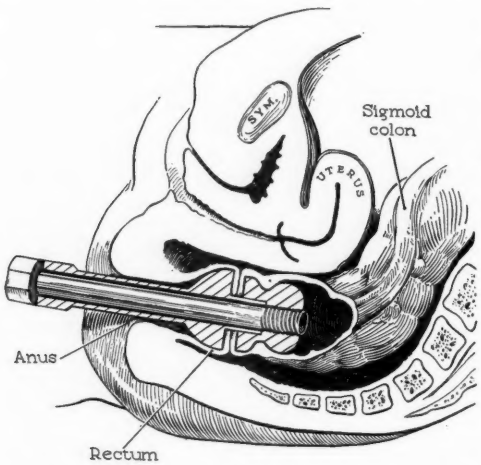


Fig. 11. Diagrammatic sketch showing instrument in place.

For these reasons, therefore, we have restricted segmental resections to lesions lying above the level of the peritoneum. The method here to be described is also highly applicable to sigmoid carcinomas for, instead of the usual V-type of excision with its limited node removal, the entire inferior mesenteric node chain may also be removed by dividing the inferior mesenteric artery at its point of emergence from the aorta with entire disregard for the lower sigmoid blood supply.

The desirability of employing a method such as this in avoiding a wet colostomy is very evident. Other applications which have occurred to us but which we have not as yet had an opportunity to explore are ano-ileal anastomosis after one stage total colectomy as used for diffuse colonic polyposis and recto-colostomy after pelvic exenteration for carcinoma of the cervix with avoidance of a wet colostomy.

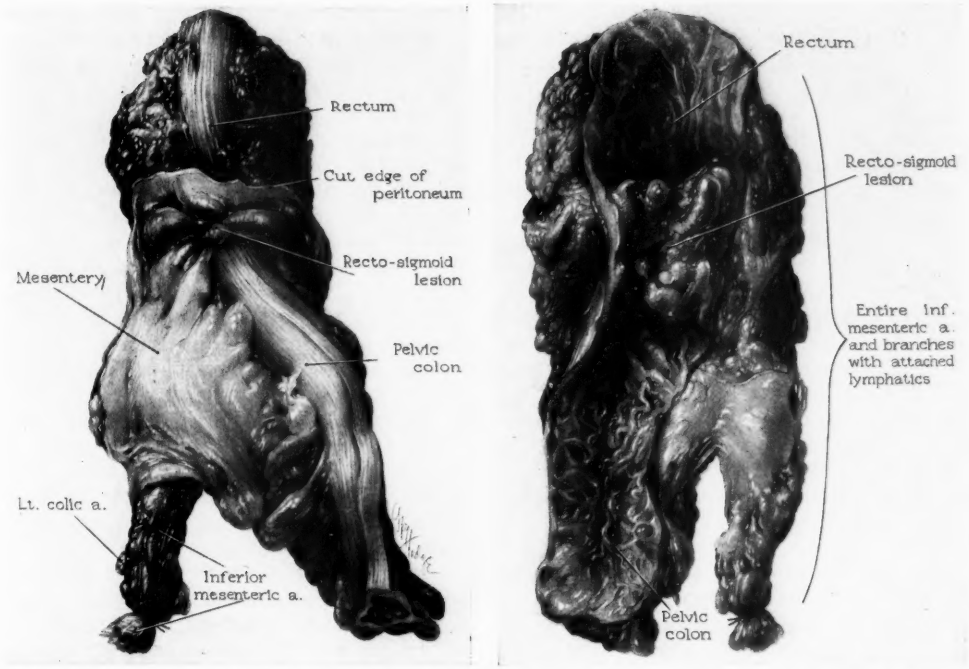


Fig. 12. A, left, Drawing of specimen with tumor just above peritoneal reflection; B, specimen opened, indicates wide margin of bowel below tumor and long node chain removal.

TECHNIQUE

No implication is intended that an instrument such as described in this article is fool proof. As with all other surgical instruments, considerable care must be exercised in its use. We have found two principles to be absolutely essential for good results. First, plenty of proximal bowel must be available. It is not sufficient to have enough bowel available to appose two ends without tension. The contraction of the longitudinal muscular coat may overcome a small amount of slack originally present with shortening of the proximal bowel postoperatively and partial separation of the apposed ends. It is our aim to have enough colon to loop on itself in the pelvic hollow and whenever this seems questionable, the splenic flexure is detached to provide a sufficient amount of coiling. Second, absolute hemostasis must be attained in the pelvis before the bowel ends are brought together. After the bowel ends have been brought in apposition, accessibility to the sacral hollow is no longer present. Our technique in the use of the instrument is shown in Figures 2 to 10 and is as follows: after preliminary bowel cleansing by use of enemas and saline catharsis, the patient is given 3 grams of sulfasuxidine every 4 hours for 5 daily doses (15 gm. daily) or approximately half that amount of sulfathalidine for 5 days preoperatively. Forty-eight hours prior to the expected date of operation streptomycin is given by mouth 0.5 gram every 6 hours for a total of 2 grams daily. The night before operation the colon is vigorously lavaged until the return flow is entirely clear and double doses of sulfasuxidine and streptomycin are given. Careful attention is paid to the patient's general condition to be considered beyond the scope of the present writing.

A left rectus incision is made so that if it becomes necessary to free the splenic flexure the incision may be extended upward and obliquely lateral to give ready access to this

portion of the colon. The lateral leaf of the parietal peritoneum is next freed mobilizing the colon (Fig. 2). The inferior mesenteric artery is ligated at its point of emergence from the aorta (Fig. 3) in order that the entire accompanying node chain may be removed. Dependence is placed entirely on the vascular arcades of the left mesocolon (Fig. 4). By transillumination of the mesentery these are divided in such a manner as to permit a good blood supply to the descending and sigmoid colons (Fig. 5) except in those occasional instances in which the arcades are not continuous. In such a situation, the splenic flexure must of course be mobilized and the transverse colon brought down. If there has been any question as to the redundancy or vascularity of the proximal bowel, final decision as to whether or not the splenic flexure need be freed should be made before the bowel lumen is cut across.

The rectum is then mobilized just as in a Miles resection down to the levator muscles (Figs. 6, 7). Care must be exercised during the dissection not to tear the muscular coats of the rectum for they are very necessary for a secure anastomosis. The superior hemorrhoidal vessels are next divided on the posterior surface of the rectum immediately below the point chosen for transection and anastomosis. A right angle clamp is placed across the rectum just above this chosen site and the division of the bowel is begun anteriorly (Fig. 8). As soon as the lumen of the rectum has been entered, any mucus or liquid stool is suctioned away and a long Allis clamp is used to grasp the bowel. The incision is continued in a circular manner placing an Allis clamp at each of the four quadrants of the bowel lumen as the division progresses to completion (Fig. 9).

A rubber shod intestinal clamp is next placed across the sigmoid several inches proximal to the site determined for its division and the bowel is cut across without the use of crushing clamps. Any bleeding points are carefully ligated following which part C of the clamp is slipped into the anus (Fig. 10, A) and that portion protruding through the anus is held beneath the drapes by an assistant. Since the rectal ampulla has a considerably larger diameter than the sigmoid in most cases, it is usually necessary to place a simple pursestring type of suture along its open end (Fig. 10, B) following which part C of the instrument is thrust gently upward impaling the bowel end on the needles. Part A is inserted into the sigmoid (Fig. 10, C), the cut end is looped over the needles, any excess bowel protruding into the lumen of the clamp is carefully cut free with a tissue scissors (Fig. 10, D) and, the position of the key being noted, parts A and C are brought together so that the key enters the slot provided for it. The two are then gently but firmly pressed together. The first few times that the clamp is used it may be helpful for the operator to contaminate one of his hands in order that he may hold the part of the clamp protruding through the anus (part C) as well as part A contained within the sigmoid. The screw stem (part B) is now inserted through the lumens of parts C and A and screwed into part A so that there is snug apposition of the two loops of the bowel. No attempt is made to crush the bowel at this point. After the bowel has been securely apposed with the instrument, we have felt it helpful and an added safety factor to use several tacking sutures of fine chromic catgut placed through the seromuscular coats of the bowel at intervals of approximately 1.5 centimeters about the circumference (Fig. 10, E).

At this point a drain is placed near the site of anastomosis. In women it has been most practical to drain through the posterior vaginal wall. In males drainage is less satisfactory and is accomplished with some difficulty by the insertion of a long clamp through the levator floor and out into the ischial rectal fossa and through a skin incision on the buttocks. The pelvic floor is then reperitonealized in any manner which is practical and simple.

The decision as to whether or not a decompressive colostomy should be accomplished must now be made. In general, we have been influenced by the cleanliness of the bowel at the time of operation. Where a partial obstruction has been present it is difficult to cleanse the bowel adequately and under such circumstances we have always done a simple decompressive loop transverse colostomy. If the bowel has been completely cleansed and is entirely free of any stool, decompression pro-

vided by the clamp appears to be adequate. If there is any question as to whether or not the bowel is entirely clean, we feel that it is safer to perform a colostomy. If a colostomy is not done the patient must be kept on a low residue diet for 4 or 5 days postoperatively. The ultimate closure of a loop type of transverse colostomy is delayed for from 6 to 8 weeks as we have indicated in a previous report (8).

The operative specimen removed by such a means as this should be adequate both in respect to width of primary removal and length of node chain (Fig. 12, A and B). In other words, no compromise whatever is made with the tumor in an attempt to save a normally functioning sphincter.

Postoperatively the patient is encouraged to lie on one or the other side. If he lies on his back he must lie flat in bed and not in Fowler's position since we fear pressure on the protruding end of the clamp. Should slight separation occur it does little harm if plenty of redundant bowel is present. If not, however, there may be a leak of stool into the presacral region. This frequently occurs in hand sutured anastomoses as others have reported. Tightening of the screw is begun 5 to 6 days postoperatively during which time the patient of course remains in bed. The clamp is usually free by the tenth postoperative day. It is simple to determine this by gentle rotation. If the clamp rotates easily, it may be removed through the anal sphincter. Following this the patient is permitted out of bed. If a decompressive transverse colostomy has been done, colon irrigations are begun through both segments of the bowel. If no decompressive colostomy has been done, the patient is put on sulfasuxidine in the same dosage as was given preoperatively which not only cuts down the bacterial content of the stool but reduces it to a consistency which makes for easy bowel movement. No enemas are given to those patients in whom transverse colostomies have not been done. The drain, either through the skin of the buttocks in the male or through the vagina in a female, is usually left in for several weeks. We have done this because some blood clot is apt to collect in the presacral space and requires a considerable period for its complete digestion and ultimate discharge.

In only one instance has a secondary closure of a partially separated anastomosis been necessary (Case 4). This was our first attempt with the clamp and ureterocolic transplants had also been done. Late strictures at the anastomotic site have not been a problem. In no instance has any become less in diameter than an examining finger, none have required dilation, and in most instances it is difficult to feel the anastomotic site.

SUMMARY

1. A new method of rectocolic anastomosis by an anastomotic clamp is discussed.

2. The method is recommended for its greater simplicity and security both in lessening initial contamination and in maintaining bowel apposition.

3. The method is not recommended for primary bowel cancer lower than the cul-de-sac level.

4. In resecting sigmoid cancer it makes easily possible the resection of the entire inferior node chain instead of a simple V-type of mesenteric removal.

5. Other types of application are suggested and the technique of its use is outlined.

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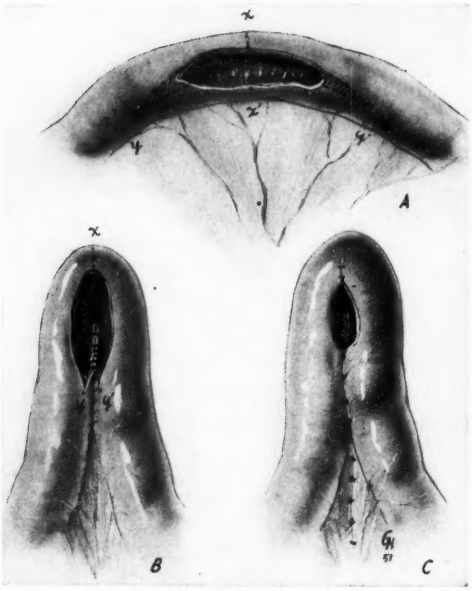

Fig. 1.

Fig. 1. Illustration of the method of closing a long defect in bowel without resection. The defect may be enlarged, as indicated in A, when it is necessary to obtain a sufficiently long stoma. B, Shows the two posterior rows of sutures in place. The bowel has been folded on itself around points x x'. C, Shows the incomplete anterior suture. The choice of suture is left to the individual surgeon. Note that a locked stitch should not be used to suture bowel, because it results in pressure necrosis under each locked loop of the suture.

Fig. 2. An extensive, ragged defect involving more than half the diameter of the bowel. The traumatized edges of the defect are trimmed as indicated in A and B. The bowel will be folded or plicated on itself around points x x'. It will be noticed that a long incision is made to point y. The points x x' are selected so as to give the full diameter of one limb of the bowel for filling in the defect at its greatest extent. C, Shows the approximation of points y and y' and the placement of the outer posterior row of interrupted sutures. The inner posterior row is placed before beginning the anterior closure. D, Shows the incomplete anterior row of sutures.

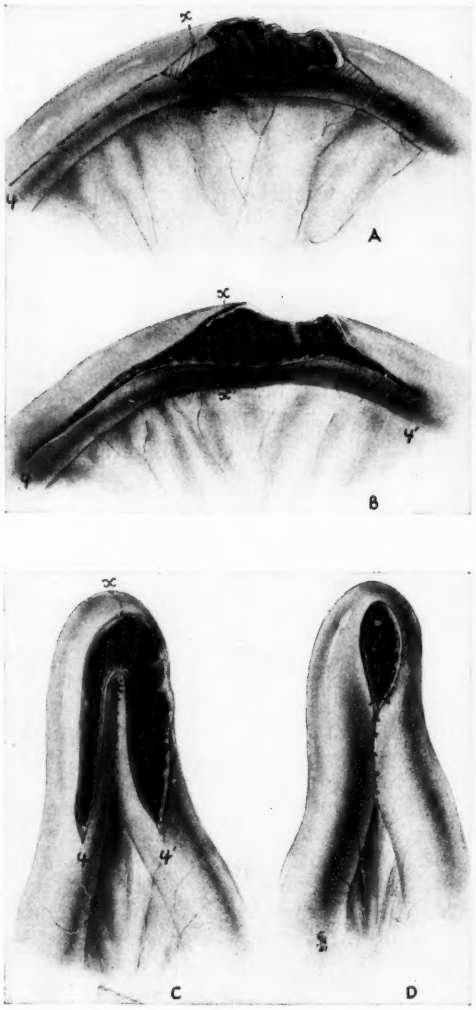

Fig. 2.

A Method of Closing Defects in Bowel.—Edgar J. Poth and Richard G. Martin.

A METHOD OF CLOSING DEFECTS IN BOWEL

EDGAR J. POTH, Ph.D., M.D., F.A.C.S., and RICHARD G. MARTIN, M.D.,
Galveston, Texas

THE procedure illustrated in Figure 1 first suggested itself to the authors when they were faced with the problem of closing a long defect in the distal ileum resulting from a gastroileostomy inadvertently done elsewhere. The defect was too long to permit transverse closure without undue tension and ordinarily would have required resection of the segment of bowel. The application of a principle recently described for bowel suture (1) gave a simple solution, and the defect was closed without resection.

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In some cases it may be necessary to enlarge the defect to provide a sufficiently large stoma, as illustrated in Figure 1 A. Another advantage of this procedure is that an adequate stoma is readily obtained without placing tension on the line of suture.

The procedure demonstrated in Figure 2 is most useful and convenient for repairing extensive traumatic lesions especially in the small bowel. This technique is applicable

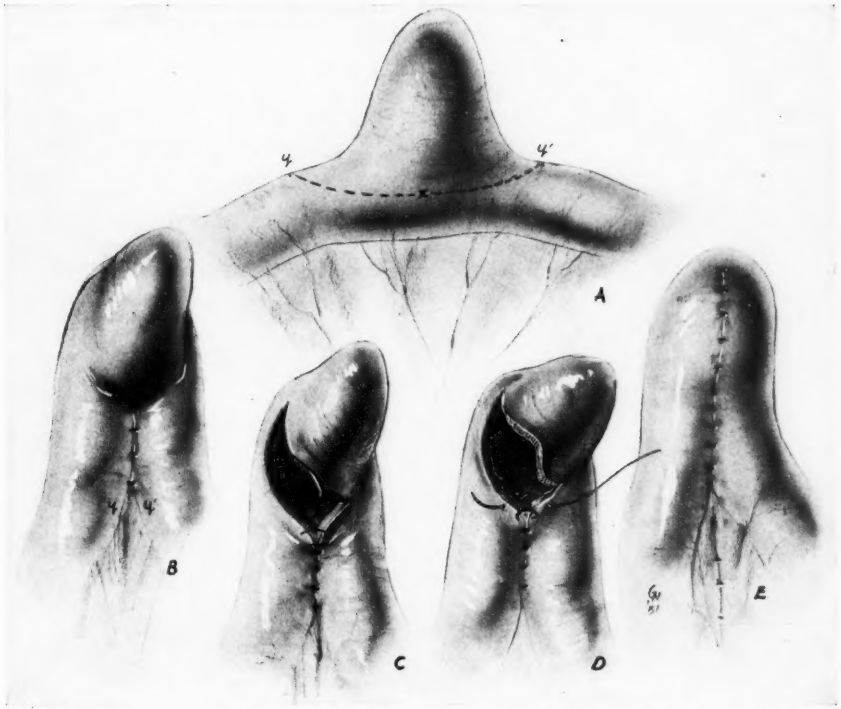

Fig. 3. An illustration of the application of the procedure to the excision of a large Meckel's diverticulum. *y y'* are approximated and a posterior row of sutures are placed in B. C, D, and E, Show the excision of the required amount of tissue together with the diverticulum to allow an adequate stoma closed without tension.

to the closure of several small defects when they are close together. The defects are connected by an incision, traumatized edges are trimmed, the resultant defect is plicated on itself and closed.

These maneuvers are so simple and clearly shown in the illustration that they require no lengthy discussion. Other applications will suggest themselves as different problems are encountered. Frequently it will not have been possible to prepare the bowel with intestinal antiseptics before operation. In these instances the entire bowel cephalad to the lesion as well as a short distal segment should be filled with 1 per cent neomycin solution, and the traumatized area should be irrigated with the same solution. It has been shown repeatedly that no bacteria can be cultured from the bowel within 45 minutes after it has been filled with 1 per cent neomycin solution. Healing of the suture line is favored by this intestinal antiseptic (2).

Neomycin is not absorbed from the gastrointestinal tract, but the amount of this antibiotic instilled or spilled into the peritoneal cavity should not exceed 0.5 gram because it is toxic when given parenterally.

The procedures herein presented have been used repeatedly and found to be quite satisfactory. In any instance in which a defect in bowel is too extensive to permit transverse closure, this plication procedure can be used.

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THE ECTOCERVIX DURING GESTATION

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IN formulating histologic diagnoses on biopsy material taken from the ectocervix during pregnancy, one sometimes faces the problem of differentiating between the benign and the malignant (4, 8, 16, 23). As references to the squamous portion of the cervix during pregnancy were sporadic (3, 15), we embarked on the study of biopsy specimens secured at various stages of gestation and at term. By the time a fairly large material had been collected by us, several essays on the uterine cervix were published (1, 2, 6, 9, 19). We hesitated therefore to present our findings until we realized that neither of the published papers comprises a sufficient number of cases for valid statistical analysis, further that these authors reach on several accounts divergent conclusions, and that we might be able to add information on a subject which apparently recently attracted the concurrent attention of several groups of investigators.

Our prime purpose was to study the microscopic architecture of the intraepithelial zones of the stratified squamous epithelium and the squamocolumnar junction at the various months of gestation and at term after vaginal delivery. It was felt that without such knowledge the clinician and the pathologist alike are handicapped in evaluating early or suspected malignant alterations of the ectocervix during pregnancy. Even in nonpregnant women such histologic diagnoses as "precancerous hyperplasia" and "intraepithelial or preinvasive carcinoma" are attended with hazard of error. Voluminous literature has accumulated on these subjects, from the diagnostic, prognostic, and therapeutic points of view.

MATERIALS AND METHODS

Punch biopsies were taken from women admitted at various months of pregnancy. Some had uncomplicated incomplete abortion, and others had excessive leucorrhea. Some were admitted for premature labor and others for full-term delivery. The tissues for study were immediately placed into Zenker-formol solution for fixation, embedded into paraffin, series of sections were made at 50 micron intervals and stained with hematoxylin-eosin and the trichrome stain.

Informations concerning our series of patients and the material studied are summarized in Tables I, II, and III. The total number of patients investigated was 75 and the total number of biopsies was 77—one woman was examined in the second month of gestation and at term, and another in the third month and again at term.

Of 75 women studied, 14 were primiparas and 61 multiparas. It should be added that curettage was performed prior to the present investigation in a total of 15 women: once in 9 cases, twice in 4 cases, and thrice in 2 cases.

OBSERVATIONS AND COMMENTS

While we had intended to study the ectocervical epithelial lining, several corollary observations were made. These will be discussed first.

Glandular changes. It might well be pointed out that cyclic changes in the endocervix have not been definitely established. While some (24, 27) take the affirmative, some 26 deny cyclic alterations. Cervical glands during gestation have been extensively investigated (3, 6, 11, 14, 19, 23, 25). It seems superfluous to elaborate on details. In summary, two major alterations present themselves during gestation: (a) a papillary intra-acinar hyperplasia characterized by long thin projections of the mucosa, and (b) a multiplicity of nuclear layers which can appear in an isolated patch of mucosa and thus present the picture of a local hyperplastic process. Ordinarily, the latter changes are not marked. In our series, not a single instance was observed in which the glandular alterations seen could be confused with adenocarcinoma. In our sections, a large number of glands and even of

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Fig. 1. Diffuse replacement of cervical stroma by dilated endocervical glands producing a struma-like pattern. ×60. Patient V. N. (No. 27), 33 years old, secundigravida; eighth month of gestation.

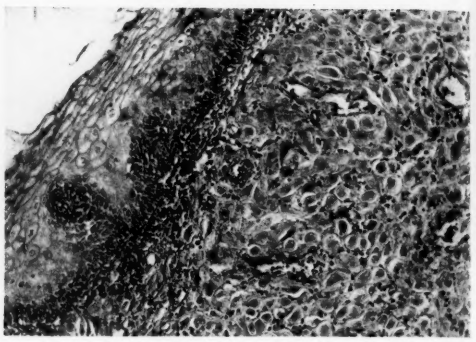

Fig. 3. Marked decidual transformation of stroma; localized basal layer hyperplasia. ×80. Patient M. D. (No. 74), 25 year old, tertigravida; 3 miscarriages, second month of gestation.

nabothian cysts was seen which remained intact even after vaginal delivery.

Multiple small nabothian cysts may so infringe upon the squamous epithelium from beneath that the lining becomes thin and eventually eroded (Fig. 1). During pregnancy, this process is facilitated by glandular activity and hypertrophy which exceeds that of the squamous portion.

Vascular alterations. Curiously, the cervical blood vessels were described but sporadically (25). In our series, the vessels were uniformly and severely congested. They contained, for the most part, large numbers of segmented cells, lymphocytes, some plasmacytes, and monocytes. In many instances, presumably due to trauma, there was an infiltration of segmented leucocytes into the stroma and also evidence of fresh interstitial bleeding. Such events may predispose to cervical erosion and vaginal bleeding. Part of the sanguineous discharge which accompanies the expression of the mucous plug during labor apparently originates from small ruptured blood vessels underlying the epithelium which desquamates because of edema and the forces of labor. The presence of intraepithelial blood and lymph vessels in nonpregnant and pregnant squamous lining alike deserves consideration, especially if one speaks of preinvasive carcinoma. Breakage through the basement membrane of the squamous epithelium does not seem always necessary for spread of the newgrowth.

Fig. 2. Marked edema of stroma and desquamation of ectocervical epithelium. ×60. Patient H. K. (No. 6), 24 years old, secundigravida; at term.

Fibrous and muscular components. With the aid of differential stains one can distinguish the muscular from the fibrous components of the stroma. The cervical stroma is

TABLE I.—DISTRIBUTION OF BIOPSY MATERIAL FROM 75 WOMEN

Month of gestation	No. of biopsies
1	1
2	13
3	14
4	8
5	8
6	7
7	5
8	6
9	10
at term	5*

*Including 2 repeat biopsies.

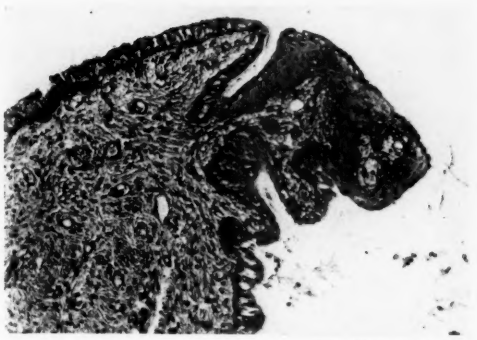

Fig. 4. Epidermization of columnar lining. ×100. Patient D. M. (No. 75), 42 years old, quartigravida; fifth week of gestation.

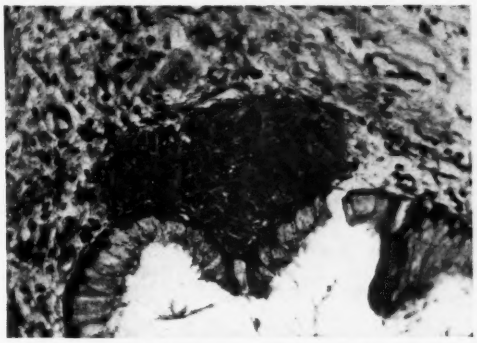

Fig. 5. Patch of squamous metaplasia. ×345. Patient L. B. (No. 15), 25 years old, secundigravida; 2 miscarriages, second month of gestation.

largely fibrous. Only an occasional cell or group of cells has the character of muscular elements. Such muscular patches are isolated and ordinarily lack continuity through several sections from the same tissue block. The bulk of the stroma, even if necessarily designated as fibrous tissue, has, in the majority of pregnant women, an extremely loose texture (15, 25). Intercellular edema was the dominant feature in most of our slides. It separated individual cells by wide spaces and compressed the cytoplasmic portions to slender, attenuated, spindle-shaped elements. This change is by no means constant (5). In about 10 per cent of our patients, edema was either negligible or absent. In these slides, the stroma had a dense, fibrous character. However, where edema is present, it seems to progress during the course of pregnancy and reaches its peak at term (Fig. 2).

Decidua formation. Decidua formation has been widely accepted as a common finding in the gravid cervix (3, 6, 7, 14, 15, 19). Even extrauterine decidua formation during gestation has been described (12, 18, 21, 22). In our series, the changes presented themselves as small groups of typical decidual cells, usually in juxtaposition to a gland. The formation of decidua was most commonly seen in the stroma at the tip of the intra-acinar glandular projections or within the stroma of the glandular projections which line the endocervical canal. In these locations the decidua was well developed and in only a single woman was there diffuse replacement of the fibrous stromal elements. It has been variously stated that decidual change occurs in 40 (23), 34.2 (3), or 22 per cent (19) of pregnant women. Minor decidua formation was observed in every cervix during gestation examined by us. One has to study multiple sections because frequently only a few cells are present at any given location and level. These nests of decidual cells though small are commonly present in large numbers, particularly beneath the columnar epithelium. The maximum decidual reaction in our series was seen in a biopsy specimen secured during the second month of

TABLE II.—AGE RANGE OF 75 EXAMINED WOMEN

Age, years	No. patients
below 20	10
21–30	46
31–40	16
over 40	3
Youngest, 17	
Oldest, 42	
Average, 27	

TABLE III.—NUMBER OF PREGNANCIES EXPERIENCED BY THE 75 WOMEN

No. of pregnancies	No of patients
I	14
II	30
III	8
IV	6
V	7
VI	3
VII	5
VIII	1
IX	1

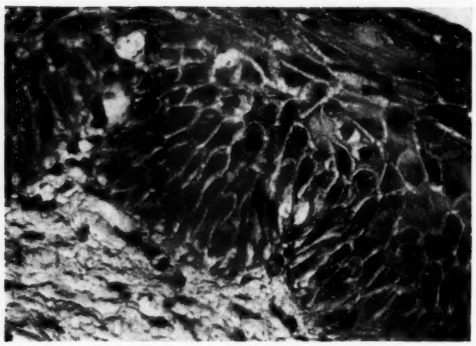

Fig. 6. Basal cell hyperactivity (note mitosis). ×495. Patient Mg. D. (No. 9), 33 years old, secundigravida; second month of gestation.

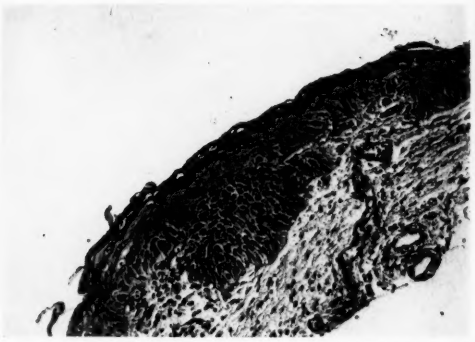

Fig. 7. Downbudding of squamous epithelium into the stroma, possibly representing the location of a destroyed endocervical gland. ×60. Same patient as in Figure 6.

gestation (Fig. 3). We gained the impression that the stroma beneath the glandular lining of the cervix is more disposed to decidua formation than the stroma beneath the ectocervix.

Decidual reaction has been reported in conjunction with cervical erosion during pregnancy (17). It has been seen in polyps during gestation (14). In some instances, deciduosis of the cervix was so marked that it simulated squamous cell carcinoma (13). It seems to us that cervical erosion is associated with excessive edema and subsequent thinning out of the epithelium rather than with decidua formation. While a dense and extensive inflammatory exudate into an area of deciduosis might obscure the architecture to such an extent that a reliable diagnosis is impossible, simple decidual change could hardly be confused with a neoplastic process

Epidermization. Epidermization of the columnar epithelium is commonly referred to as metaplasia. Initially, there is an insidious squamous cell proliferation just below the basement membrane of the glandular epithelium. Eventually, this process replaces the epithelium of cervical glands and their lumina and forms in the last analysis an extensive displacement of the destroyed glandular latticework by squamous cells.

The frequency of such alterations in pregnant women has been variously reported as 18 (19), 18.7 (3), about 50 (1, 15), and as 86 per cent (2). In 44 per cent of cases, stratification of the columnar epithelium was seen (2). Our own figures approach the lowest ones cited: Epidermization was observed in 12 out of 75 women, or in 16 per cent of the series (Figs. 4 and 5).

In the normal organ there is a fairly distinct line of demarcation between the squamous and the glandular epithelium. It is at this zone of epithelial interplay that squamous metaplasia is seen. During and after pregnancy, as during fetal life of the female, this zone is the site of dynamic changes. During gestation there is a rapid and extensive proliferation of the glandular portion. The squamous part presents much less proliferative activity. In a way, the process is the antithesis of epidermization; glandular replacement of the squamous lining takes place.

The squamous lining. The alterations of the ectocervical epithelium consist of: (1) irregularity of basal cell nuclei, (2) disturbed polarity of the basal cells, (3,4) variation in the shape of cells and nuclei, (5) increased number of nucleoli, (6) mitotic activity, (7) budding of the epithelium, and (8) thickening of the basal cell layer. There are great discrepancies in the various reports (1, 2, 3, 6, 15, 19) as to the frequency of these phenomena during pregnancy.

Basal cell hyperactivity as characterized by disturbed architecture, cellular and nuclear pleomorphism, and by mitotic activity has been observed as reversible phenomenon, without serious consequences. According to

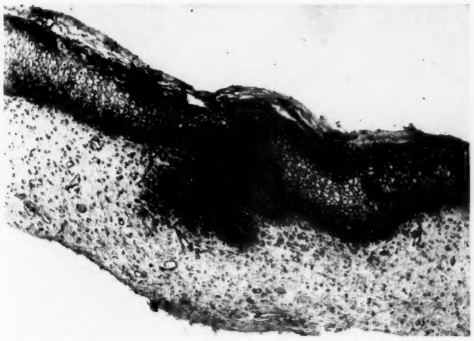

Fig. 8. Downbudding of squamous epithelium. ×80. Patient J. P. (No. 22), 19 years old, primigravida; first month of gestation. (Postpartum biopsy; no alterations.)

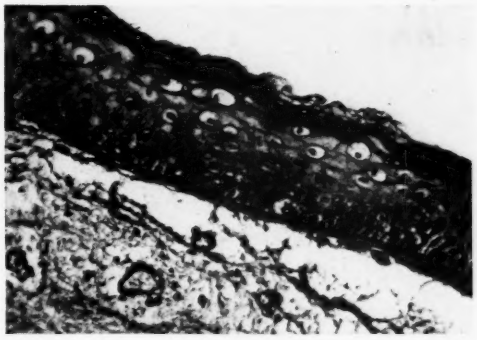

Fig. 9. Squamous epithelium in a case of hydatiform mole. ×345. Patient D. H. (No. 38), 22 years old, primigravida (miscarriage); sixth month of gestation.

three different authors, these alterations occur in 6 per cent of 50 (19), in 12.5 per cent of 32 (3), or in 64 per cent of 22 pregnant women (2). In our series of 75 patients, increased basal cell activity was seen but 4 times (3 per cent) (Fig. 6). Even where present, such hyperplastic process never extended through the entire thickness of the epithelium. A breakdown into the components of the microscopic picture and calculation of the percentage of incidence of supernumeric nucleoli, mitosis, etc., fails to add information or to reveal any deviation from their frequency in nonpregnant women of comparable age.

Ingrowth of the squamous epithelium into the stroma has been reported as occurring in 64 per cent of 22 patients (2). This figure was arrived at by analysis of a small group of selected patients. In our series of 77 biopsies from 75 women, downbudding of the squamous epithelium was observed 5 times (6.5 per cent of biopsies, or 6.6 per cent of patients) (Figs. 7 and 8). It was felt that such processes represent the end result of a previously active squamous metaplasia and that the configuration of these buds is predetermined by the outline of any particular replaced endocervical gland.

Thickening of the squamous stratified epithelium has been mentioned by all students of the ectocervix during pregnancy. It was seen in the vast majority of our slides. Most investigators hold thickening of the basal layers responsible for the thickening of the lining as such. It seems to us that the epithelial hypertrophy is not due to basal cell hyperactivity but rather to an increase in the size of the individual cells comprising the intermediate layer of the squamous epithelium. Nuclear enlargement in the midzone has been described in 73 per cent of pregnant women (2). Beside the increase in nuclear and cellular size, intercellular edema of the midzone is responsible for the increased width of the squamous lining.

Only one author (28) ascribes hypertrophy of the squamous part to the action of luteal hormone and the hypertrophy of the columnar portion to estrogen. Simultaneous administration of both hormones would result in reaction of both types of cervical epithelium. Commonly, estrogenic stimulus in pregnancy is being offered as explanation of the basal cell hyperactivity and for the hypertrophy of the squamous lining (1, 3, 19). Cervical hyperkeratosis and parakeratosis were absent in patients who died of eclampsia with lessened estrogen production (6). On the other hand, thickening of the ectocervical lining was greater in early pregnancy than in late although the amount of estrogen usually increases progressively from the seventeenth week of gestation till term (16).

In view of this controversy, it was of interest that in 1 of our patients, in whom biopsy was taken 3½ months after onset of her first gestation and who had grossly and histologically proved hydatiform mole, the squamous

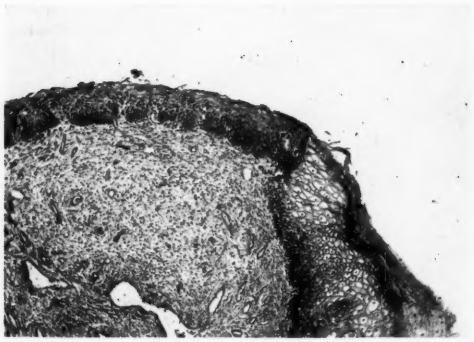

Fig. 10. Sharp line of demarcation between normal and abnormal sectors of squamous epithelium. ×60. Patient R. U. (No. 49), 24 years old, quartigravida; 1 miscarriage and curettage; eighth month of gestation.

Fig. 11. Same as Figure 10. ×345.

lining was intact and thin throughout (Fig. 9). No determination of estrogen was performed but theoretically its amount should be abnormally high. There seem to be exceptions to the rule that hypertrophy of the ectocervical lining is linked with the increase of estrogen production during gestation.

DISCUSSION

This study was carried out to answer the question as to whether or not "intraepithelial carcinoma" of the cervix might arise secondarily to the varied stimuli during gestation. This question has been answered only in so far as in a series of 75 patients not a single instance of intraepithelial carcinoma or of alterations even remotely resembling neoplastic activity was seen.

In 2 of our patients there was a sharp line of demarcation within the squamous epithelium separating an abnormal sector from a normal one (Figs. 10 and 11). Such line of demarcation is commonly seen at the edges of leucoplakia rather than at the edges of intraepithelial carcinoma. The process may in fact represent a phase of leucoplakia transformation. These alterations were not grossly visible at the time biopsies were taken.

Experimentally, atypical growth of the cervical epithelium of the monkey was produced by prolonged injection of estrogen together with repeated trauma to the cervix (20). Such experimental alterations resemble malignant cervical neoplasm of women (10). In view of such studies, it seemed advisable to compare the incidence of hyperplastic processes in primiparous women with those observed in multiparous, and further to investigate such alterations in women who had had miscarriages and if curettage had been done prior to present examination. As far as one may be permitted to draw conclusions from a study of 75 patients, we could not correlate any of the alterations with either the age of the patients, the number of pregnancies or miscarriages, or with previous curettage. This was true not only of alterations of the squamous lining proper but also of the incidence and extent of epidermization, of decidua formation, stromal edema, and the like.

Correlation of microscopic observations with the month of gestation revealed nothing but progress of stromal edema and an increase in number and size of glands in the course of gestation. Epidermization, downbudding of the squamous lining, and basal cell hyperactivity were observed already during the first month of gestation in a primigravid, 19 year old women while another patient with comparable alterations was 42 years old, gravid for the fourth time, and in her second month of pregnancy. These observations agree with a report (2) on multiple alterations in material obtained during the first 5 months of gestation but differ from one (15) of progressive changes in microscopic structure and appearance of certain formations at given stages of pregnancy.

Any one structural change observed during gestation can be seen in cervical biopsy mate-

rial from nonpregnant women. The alterations observed in the ectocervix hardly exceed analogous changes in tissues from nonpregnant women, either in frequency or in degree. In the pregnant and nonpregnant woman alike, hyperplastic and hypertrophic processes might occur in a cervix which appears normal to gross inspection. This holds true of leucoplakia and of intraepithelial carcinoma, as well. The diagnosis of preinvasive, intraepithelial carcinoma or of precancerous lesion should be made with caution, at any time.

SUMMARY

1. Seventy-five patients at various stages of pregnancy were studied with regard to morphologic appearance of cervical structures.

2. Analysis of 77 cervical biopsies from 75 women confirms observations of other investigators but differs as to the incidence of alterations.

3. Alterations of the ectocervix are described and their theoretical and practical implications are discussed.

4. None of the alterations found is characteristic for pregnancy or occurs in pregnancy significantly more often than outside of pregnancy.

5. Correlation of structural changes with age, frequency of pregnancies, duration of gestation, miscarriage, and curettage cannot be established.

6. Hypertrophy of the ectocervical lining is ascribed to midzone changes rather than to basal layer activity.

7. On the basis of observations in an instance of hydatid mole, the interrelationship between estrogen production and squamous cell activity is discussed.

8. The possible relation to leucoplakia is mentioned on the basis of findings in 2 patients.

9. No instance of intraepithelial carcinoma or alterations suggestive of neoplasm was observed in the material studied.

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THE EFFECT OF BANTHINE, VAGOTOMY, AND SUBTOTAL GASTRECTOMY UPON PANCREATIC SECRETION

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THE purpose of this investigation is to study the effects that banthine, vagotomy, and subtotal gastrectomy have upon pancreatic secretion.

Theoretical and experimental evidence indicate that these procedures will depress pancreatic secretion. Subtotal gastrectomy should restrict the humoral phase of pancreatic secretion by reducing gastric acidity and hence secretin formation. Banthine and vagotomy should eliminate the neurogenic control of pancreatic secretion that traverses the vagi, and in addition they should depress the humoral phase by reducing the gastric acidity.

Depression of pancreatic secretion is advantageous for the treatment of pancreatitis. In the treatment of acute pancreatitis, gastric suction, limited oral intake, and restriction of glucose are used in an attempt to place the pancreas at rest. From the etiological standpoint, depression of pancreatic secretion is also desirable according to the theory that pancreatitis is caused by obstruction of the outflow of pancreatic juice. The experimental work of Rich and Duff, Lium, Popper, and others (2, 13) support this concept. The clinical association of attacks of pancreatitis to food ingestion and alcoholic bouts favors this theory, since both stimulate pancreatic secretion.

There have been a few reported cases of chronic relapsing pancreatitis treated by vagotomy and subtotal gastrectomy (9). Vagotomy with gastroenterostomy has also given encouraging results in some instances. Preliminary reports indicate that banthine is beneficial in the treatment of acute pancreatitis (11). Recently Annis and Hallenbeck, working with dogs, demonstrated that banthine in large doses reduced pancreatic secretion.

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METHODS

Mongrel dogs of both sexes weighing 6 to 10 kilograms were used. The animals were anesthetized with intravenous nembutal and all operations were performed with aseptic technique. All dogs were fed a standard commercial dog diet during the postoperative period. The pancreatic secretions were collected by two methods. In one group of animals the major pancreatic duct was cannulated. In another group of animals the method of Dragstedt, Haymend, and Ellis was used, creating an isolated pouch of duodenum with only the pancreatic ducts entering it. The effect that banthine, vagotomy, and subtotal gastrectomy have upon pancreatic secretion was studied by these two methods and the results compared. The first type of preparation was practical for acute studies, while the other method allowed observation for prolonged periods after the animals had recovered from the effects of the operations.

The pancreatic duct was cannulated in 12 dogs. Eight dogs were satisfactory for study, but in the other 4 dogs pancreatic juice failed to flow from the cannula or stopped flowing before adequate samples could be obtained. Collections were made at 10 minute intervals. Samples were collected until 2 consecutive ones were of equal volume, and then 2 samples were collected during each test. After a rest period, additional tests were made upon the same animal. Samples were thus collected before and after each test so that changes observed were adequately controlled. This eliminated comparisons of one animal to another. The first sample of each collection period was discarded after being measured. This served to wash out the cannula. The specimens were measured and analyzed promptly for amylase content. The common duct was occluded in all animals to eliminate the influence of bile, and the pylorus was occluded except when it was intended for acid to enter the duodenum.

Nineteen dogs were prepared after the method of Dragstedt and associates, but only 9 of these lived long enough for satisfactory study. The isolated duodenal pouch was cannulated with a catheter from which collections were made. Pancreatic secretions were allowed to return to the animal by connecting this catheter to another which was placed in the jejunum. In this way it was possible to keep these preparations alive for several months. Three-fourths gastric resection was performed in 4 of the satisfactory animals at the time of the original procedure. The other 5 satisfactory animals had only the pyloric antrum resected. Eight of the 10 animals that died succumbed before the ninth postoperative day; 2 of pneumonia and distemper, 4 of peritonitis secondary to a leak about one of the catheters, and 2 from breakdown of the transplanted common duct anastomosis. Two developed an ileoileal intussusception for no apparent reason several days after operation.

Collections were made at 2 hour intervals from the isolated duodenal pouches. These specimens were contaminated with succus entericus, therefore volume determinations of pancreatic juice collected by this method were not felt to be accurate. Total amylase content of the specimens was a significant determination because it was not altered by the changes in volume due to the succus entericus. Amylase contents of the 2 hour specimens in different animals varied between 5,000 and 18,000 units. For clarity, changes in amylase content following the tests are reported in per cent. It should be remembered that amylase levels are 10 times higher in the dog than in man. Amylase determinations were made on all specimens promptly after collection. The method of Smith and Roe was used.

RESULTS

Banthine. This drug was tested in 4 of the dogs in which the pancreatic duct had been cannulated. The common duct and pylorus were occluded. Banthine was administered intravenously in doses of 0.3 to 0.5 milligram per kilogram. This dosage caused dilatation of the pupils but no appreciable change in blood pressure. Samples of pancreatic juice were collected after the administration of this drug, and then after a rest period priscoline was tested. It was given in doses of 10 to 15 milligrams intravenously. Banthine reduced the volume of flow in all 4 animals an average of 19 per cent (11 to 28 per cent) and reduced the amylase content per milliliter by approximately the same amount. The amylase content per specimen was decreased more than the concentration per milliliter since the volume had been reduced (Tables I and II).

TABLE I.—VOLUME SAMPLES FROM PANCREATIC DUCT

Dog No.	Control c.c.	Banthine c.c.	Priscoline c.c.
1	.45	.40	.42
2	1.20	.91	1.02
3	.28	.21	.30
4	.86	.74	.86
Average	.70	.57	.65

Priscoline had no appreciable effect upon the volume of flow or upon the amylase content of the specimens (Table I).

In the duodenal pouch preparations, banthine was given in 0.5 milligram per kilogram doses intravenously at 2 hour intervals for the desired length of time. Results were considered satisfactory if 2 consecutive control collections had approximately the same volume and if the volumes returned to control levels following the administration of the drug. The volume in the 2 hour collections from the pouches decreased 10 to 40 per cent after administration of banthine. It has been pointed out that volume determinations in this type preparation may not be accurate since collections are contaminated with succus entericus.

TABLE II.—AMYLASE CONTENT OF PANCREATIC SECRETION

Dog No.	Control		Banthine	
	units/c.c.	units/ specimen	units/c.c.	units/ specimen
1	1,150	516	1,020	408
2	2,150	2,580	1,810	1,640
3	575	161	522	121
4	752	647	727	538
Average	1,159	976	1,027	577

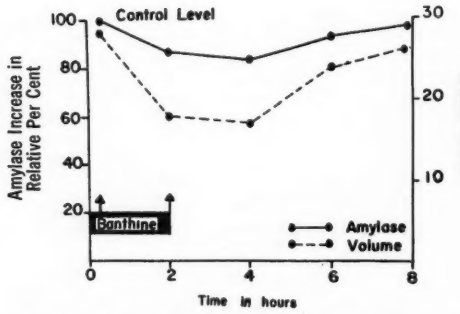

Fig. 1. Effect of banthine on volume and amylase content of pancreatic secretion.

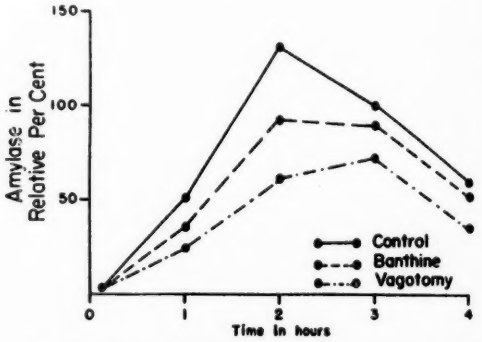

Fig. 2. Effect of banthine and vagotomy on amylase content of pancreatic secretion in response to feeding.

The amylase content per specimen decreased 15 to 20 per cent. This response was observed on at least 3 occasions in the 5 animals studied. There was no apparent summation effect when repeated doses of banthine were given, and the effect seemed to last about 2 hours. Both volume and amylase concentrations of the specimens returned to plus or minus 5 per cent of control levels within 4 hours following the last injection of the drug. Figure 1 is a graphic representation of a typical response of 1 of the animals to banthine.

An attempt was made to determine the effect banthine might have upon amylase secretion after feeding an animal a meal of standard dog diet. The control amylase curve in an animal was established by collecting specimens from the isolated duodenal pouch at hourly intervals for 4 hours following feeding. The following day, after control samples were taken, the animal was given 0.5 milligram per kilogram of banthine intravenously before feeding and collections repeated in the same fashion. The response was not as great. The highest increase in the amylase level was 90 per cent in the 2 hour specimen, while in the control test it was 130 per cent in the 2 hour specimen (Fig. 2). Annis and Hallenbeck found similar changes with banthine after feeding their animals horse meat. With larger doses they found a greater depression in pancreatic secretion.

Vagotomy. The results were more striking following vagotomy than banthine administration.

Bilateral transthoracic vagotomy was performed in 4 animals in which the pancreatic duct had been cannulated. The volume of pancreatic secretion fell an average of 34 per cent (28 to 46 per cent) and the amylase content per specimen fell 48 per cent (28 to 64 per cent) (Tables III and IV). The effect of vagotomy apparently is directly upon the pancreas rather than secondary to reduction in gastric acidity, for this response was observed when the pylorus was occluded. In 2

TABLE III.—VOLUME SAMPLES FROM PANCREATIC DUCT

Dog No.	Control c.c.	Vagotomy c.c.
2	1.20	.79
4	.86	.60
5	.73	.52
6	.56	.31
Average	.86	.56

TABLE IV.—AMYLASE CONTENT OF PANCREATIC SECRETION

Dog No.	Control		Vagotomy	
	units/c.c.	units/ specimen	units/c.c.	units/ specimen
2	2,150	2,580	1,190	940
4	752	647	524	314
5	512	374	520	270
6	1,810	1,014	1,670	518
Average	1,306	1,156	961	511

other animals bilateral sympathectomy was performed. There was no apparent effect upon the volume or amylase content of specimens.

In the duodenal pouch dogs, vagotomy was performed in 2 animals 14 days following the original procedure. One of these animals was observed for 20 days following vagotomy, but unfortunately the results from the other animal cannot be considered valid for it developed severe vomiting following vagotomy and expired 6 days later. In the animal observed, the amylase concentrations per 2 hour specimen fell 50 per cent for the first 2 days following vagotomy, then gradually increased, and from the seventh to the twentieth day remained about 20 per cent below control levels (Fig. 3).

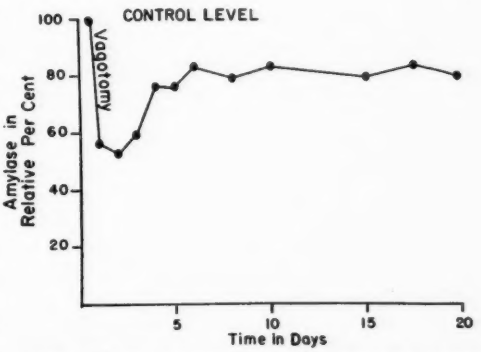

Fig. 3. Effect of vagotomy on amylase content of pancreatic secretion.

This type of response following vagotomy was observed by Crider and Thomas. In their experiments, the volume output of pancreatic juice fell 40 to 50 per cent immediately following vagotomy and then later returned to near normal, but with some persistent decrease in volume. They felt this represented that the local reflex centers were able to recover and function independently following vagotomy. The results of this study and theirs are similar in that there is a marked decrease in pancreatic secretion immediately following vagotomy and that there is a 20 per cent permanent decrease. Vagotomy can be expected to have an even greater effect in cases of pancreatitis when there is excessive pancreatic stimulation traversing the vagi.

The effect of vagotomy upon amylase secretion in response to feeding was determined in 1 animal 10 days after the nerves were sectioned. There was a delay before the amylase level increased. The highest increase was 60 per cent in the 3 hour specimen as compared to a control increase of 130 per cent in the 2 hour specimen. This delay in response to feeding could be due to the abolition of the cephalic phase of pancreatic secretion or could be caused by delayed gastric emptying as a result of vagotomy. The effect following vagotomy was greater than that found in the same animal when banthine was given (Fig. 2).

Subtotal gastrectomy. The influence of subtotal gastric resection on pancreatic secretion was difficult to determine.

In 2 animals with the pancreatic duct cannulated, it was attempted to determine the effect upon pancreatic secretion when the pylorus was opened and closed. This gave inconsistent results. Previous observers have noted this. Scott and associates found that pancreatic secretory rates were not correlated with the presence of intragastric acid. Occasionally they found that gastric juice (*p*H 1.0 to 1.5) was present in the stomach in quantities up to 200 milliliters without initiating pancreatic secretion.

By using an indirect method of study of injecting 0.1 normal hydrochloric acid into the duodenum, an increase in pancreatic secretion was found. This has been demonstrated previously by many workers. When acid was injected into the first loop of jejunum, an increase in the secretion also occurred, but was about 50 per cent that seen when the acid was injected into the duodenum.

This would indicate that if a gastric resection were performed for pancreatitis, a long anticolic loop would be preferable since hydrochloric acid remaining after gastrectomy would pass into an area where the secretin response is not as great as that found in the duodenum. Kolouch demonstrated this by producing pancreatic fistulas in dogs and then performing three-fourths gastric resections. In 1 group of animals he used a short afferent jejunal loop and in the other a long jejunal loop. The comparisons of pancreatic secretion before and after resection showed a reduction in volume in both groups and a more pro-

nounced reduction in the group with a long afferent loop.

In the duodenal pouch dogs, 4 animals were prepared in the standard fashion except that three-fourths of the stomach was resected. Volumes obtained from the pouches in these animals were less than those in animals in which the stomach was not resected. The amylase content was about the same.

These results are difficult to evaluate because there may be considerable difference between one dog and another. For this reason no definite conclusions can be made regarding the effect of gastric resection in pouch dogs.

Effect upon the sphincter of Oddi. The common duct was cannulated and connected to a saline manometer. Observations were made in 2 animals which were consistent. Control pressure readings were 150 and 180 millimeters of saline respectively. When the vagi were stimulated in the neck the column of saline would not fall in the manometer when it was elevated to 300 millimeters, but would fall to control levels soon after stimulation ceased. The same occurred when the sympathetic chains were stimulated in the chest. Administration of banthine or priscoline, and cutting the vagi in the chest just above the diaphragm did not alter the resistance of the sphincter. Removing or blocking the sympathetic chains low in the chest with procaine reduced the pressure in the manometer in both animals. In 1 dog the right chain was removed first and in the other the left chain was blocked first. In each there was a fall of 40 millimeters of saline. When the other chain was excised or blocked, the sphincter resistance was 80 and 100 millimeters of saline respectively. The saline manometer level fell to zero when 2 per cent procaine solution was applied to the sphincter by injecting it into the cannula, which reaction indicated that vagotomy and sympathectomy do not completely paralyze the sphincter.

SUMMARY

The effects that banthine, vagotomy, and subtotal gastrectomy have upon pancreatic secretion were studied in dogs.

Banthine was found to decrease by 20 per cent the volume and amylase content of pancreatic secretion.

Immediately following vagotomy, the volume and amylase content of pancreatic secretion was depressed by 30 to 50 per cent. There was a 20 per cent permanent depressing effect. This response was not secondary to reduction of gastric acidity.

The effect of subtotal gastrectomy upon pancreatic secretion was variable.

Banthine and vagotomy did not alter the resistance of the sphincter of Oddi. Bilateral transthoracic interruption or procaine block of the sympathetic chains decreased sphincter resistance. The sphincter of Oddi was paralyzed when novocain was applied directly.

Although these results in dogs suggest methods of therapy in pancreatitis, the final determination of their value in humans remains to be proved.

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PANCREATIC INJURIES

An Experimental Study

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EFFECTS of various experimental injuries to the pancreas of the dog have been reported in the earlier literature (1, 2) and have been confirmed and extended recently (4, 5). Transection of the main pancreatic duct, or lengthwise incision of the duct and of its two main branches was of no serious consequence in the animals starved before and after operation. The opened pancreatic ducts generally were found sealed by omentum or by a loop of bowel. However, marked intra-abdominal fat necrosis was observed if the dogs had been fed preoperatively or postoperatively or if cholinergic drugs had been administered postoperatively.

More extensive injuries to the pancreas, such as crushing with Payr clamps or complete transverse tearing of the gland, were not followed by fat necrosis or other severe abdominal disease, independent of whether the animals had been fed before and after the operation. In every case, the traumatized portion of the pancreas was found to be covered by firmly adherent omentum (4, 5).

The result of the first series of experiments demonstrated that, following injuries to the pancreatic duct, plugging of the open duct by omentum or by loops of intestine prevented spilling of pancreatic juice into the peritoneal cavity. Obviously this was possible only if there was very little or no pancreatic secretion, because sufficient secretory pressure would have prevented sealing of the open duct by omentum.

Pavlov has pointed out that following handling of the pancreas, its external secretion is suppressed for some time. Thus, we may surmise that an arrest of external pancreatic secretion occurred in our experimental animals, which allowed plugging of the open duct by omentum. After a few days the plug had become adherent and was able to resist the secretory pressure of the recovered pancreas. At this point the healing process terminated as far as prevention of spilling of pancreatic juice was concerned. However, if during the first few days after the pancreatic injury, pancreatic secretion was stimulated by feeding or by cholinergic drugs, the secretory inhibition was overcome and plugging of the open duct was prevented by the forthcoming pancreatic secretions. Subsequently, pancreatic juice spilled into the peritoneal cavity and intra-abdominal fat necrosis developed.

The unexpected observation that more extensive injuries like crushing or tearing of the pancreas were not followed by acute intra-abdominal lesions, regardless of whether the dogs were fed or starved, was explained by the author (5) as due to a more drastic depression of external pancreatic secretion by more extensive injuries. This secretory depression apparently resisted stimulation by food, thus preventing interference with the plugging mechanism.

A further analysis of this problem, in which an attempt was made to approximate clinical conditions is presented in the following.

EXPERIMENTS

Fifty healthy mongrel dogs of both sexes of from 8 to 16 kilograms in body weight were subjected to aseptic operations under pentobarbital sodium anesthesia. The dogs did not receive any medication with the exception of secretin in a number of animals.

In 21 animals the pancreas was torn as described previously (5). In 2 of these dogs approximately 3 ounces of fresh blood were left in the peritoneal cavity, in 2 dogs, 3 milliliters of fresh sterile dog gall-bladder bile was injected into the abdomen before the closure

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of the incision, and in 2 dogs a dime sized portion of the free surface of the gall bladder was excised. In the other 15 dogs of this group, pancreatic secretion was stimulated vigorously; 2 dogs who had been trained before operation to take 20 per cent ethyl alcohol in their drinking water were given alcohol in water immediately before and after the operation. Three dogs received 5 doses of 2 milligrams of secretin each at hourly intervals beginning 48 hours after the operation; 2 dogs, 1 dose of 40 milligrams of secretin each, 2 hours after the operation; 3 dogs, 1 dose of 40 milligrams of secretin each, 24 hours after the operation; and 2 dogs, 1 dose of 40 milligrams of secretin each, 48 hours after the operation.

The 2 dogs with the opened gall bladder died within 8 hours after the operation, and autopsy showed diffuse biliary peritonitis but no fat necrosis. All other dogs survived and were sacrificed between the third and the tenth postoperative day. In all animals the omentum was firmly adherent to the pancreas. In a few, the region of the tear contained a small amount of well walled off cloudy hemorrhagic fluid. Only 1 dog, who had received 3 doses of secretin 48 hours after the operation and who was sacrificed on the eighth postoperative day, showed marked intraperitoneal fat necrosis. None of the other animals showed any fat necrosis.

In the next group of 19 dogs, the pancreas was torn in 17 and crushed in 2 of the animals. In all dogs an approximately 2 inch long piece of omentum was devitalized by tying it off with a silk ligature. Eight dogs died between the second and the sixth postoperative day, and the other 11 were sacrificed between the third and the twenty-second postoperative day. The majority of dogs developed intraperitoneal abscesses, usually peripancreatic, with necrotic tissue in the center, which caused the death of some dogs, but which were asymptomatic in the other animals. However, only in 1 of these 19 dogs extensive intraperitoneal fat necrosis was found, while 3 other dogs had a moderate degree of fat necrosis.

In 4 control experiments the pancreas was not injured, but a piece of omentum was devitalized by ligation as described previously. The dogs appeared normal, and they were sacrificed on the fourteenth day after operation. In all 4 dogs an abscess had formed around the isolated piece of omentum; however, the abscess was much smaller than the mass and abscess formation found in the dogs with additional injury to the pancreas.

In another group of 6 dogs, the pancreas was torn as usual. In addition, the tail of the pancreas was isolated and deprived of its blood supply by transection between 2 ligatures of heavy silk placed between body and tail and by ligation of remaining blood vessels. Two of these dogs died on the third and eighteenth day respectively and showed extensive intraperitoneal fat necrosis. Two dogs were sacrificed on the nineteenth postoperative day and showed only induration and atrophy of the pancreas. The remaining 2 dogs which were sacrificed on the sixth postoperative day likewise did not show fat necrosis but inflammatory masses around the ligated, partly necrotic, partly hemorrhagic tail of the pancreas, very similar to the inflammatory masses with abscess formation seen in the previous series of experiments with ligation of a piece of omentum.

The observation previously mentioned that preoperative and postoperative feeding did not influence the healing process of the torn pancreas (5) was corroborated in these experiments. Some of the dogs were fed before and after the operation, but this did not affect the results and, therefore, this procedure was not mentioned in the experiments reported above.

DISCUSSION

These experiments show that the presence of moderate amounts of blood or bile in the peritoneal cavity did not alter the uneventful course following tearing of the pancreas. The attempt of extensive drainage of bile into the abdomen through a good-sized hole in the wall of the gall bladder could not be evaluated in this respect because it led to fulminating, fatal, biliary peritonitis.

The experiments showed further that the inhibition of pancreatic secretion due to severe injuries to the pancreas will, in the immediate postoperative period and during the first few postoperative days, resist such vigorous stimu-

lation as injection of secretin or administration of alcohol. The latter is regarded as a potent stimulus of pancreatic secretion and has the advantage that, in previously trained dogs, it can be started postoperatively at a time when the animals still refuse food.

It was furthermore evident that the incidence of fat necrosis following tearing of the pancreas was not appreciably increased by the presence of necrotic omentum in the abdominal cavity or even by ischemic necrosis of part of the pancreas. Although necrosis of part of the pancreas is accompanied frequently by fat necrosis, this occurred in only 2 out of 6 dogs with a torn pancreas.

The experiments demonstrated that, in the presence of pancreatic injury, the inflammatory reaction around a devitalized portion of omentum was markedly increased.

If we assume that the reaction of the pancreas to injuries is the same in man as in the dog, we may expect that in man, following severe injuries to the pancreas or following major surgery on the pancreas, a drastic inhibition of pancreatic secretion will occur, which will resist stimulation by rather potent stimuli and which will permit an efficient sealing off of the pancreatic wound. This may explain why extensive surgical procedures on the pancreas are tolerated relatively well, while insignificant surgical or accidental injuries are followed not rarely by extensive intra-abdominal fat necrosis.

Apparently, the avoidance of stimulation of external pancreatic secretion by food or medication, as recommended for pancreatic injuries and after surgery on the pancreas (4), is by far not so important in severe injuries and following extensive pancreatic surgery as it seems to be after minor injuries, after minor surgical interventions on the pancreas, or after accidental section of a pancreatic duct, when profound secretory inhibition, resistant to pancreatic stimulation, will not occur.

The finding of resistance of the pancreas to stimulation after extensive pancreatic injuries is of practical importance because in the seriously ill patient the complete avoidance of pancreatic stimulation may be a difficult problem. It seems plausible that, following extensive surgery or severe injuries of the pancreas, moderate stimulation of external pancreatic secretion such as is produced by morphine or by intravenous solutions of glucose or of amino acids will probably be of no consequence, while it should be avoided strictly in cases of acute pancreatitis, in suspected lesser traumatic or surgical injuries, or after biopsies of the pancreas.

Furthermore, the process of self-healing of wounds of the pancreas by sealing with omentum or with loops of intestine apparently is not interfered with by moderate intraperitoneal bleeding or by intraperitoneal leakage of moderate amounts of bile.

It is generally regarded as permissible in abdominal surgery to leave in the peritoneal cavity a tag of omentum which has been deprived of its blood supply and, in fact, every ligature of omentum creates a stump of devitalized tissue. This, as a rule, is of no consequence and is tolerated so well that free grafts of omentum have been recommended for various purposes, such as the closure of a perforated peptic ulcer (6). The presence of necrotic tissue in the abdominal cavity, or even of ischemic necrosis of part of the pancreas does not seem to interfere with the sealing of a wound of the pancreas. However, the afore-mentioned aggravating effect of pancreatic injury on the mass formation around necrotic tissue seems to suggest that careful avoidance of leaving any devitalized tissue in the abdomen might prevent untoward inflammatory reactions in cases with surgery of the pancreas.

It is realized that bacterial contamination probably played a role in the formation of inflammatory masses around necrotic tissue. However, also in clinical surgery of the pancreas bacterial contamination will very often be unavoidable and it will occasionally not respond to antibiotics, thereby creating the conditions of our experimental observations.

SUMMARY

Experimental injuries to the pancreas were studied under conditions in which an attempt was made to duplicate possible clinical circumstances.

It was found that the presence of moderate amounts of blood or sterile bile in the peri-

toneal cavity did not alter the uneventful course following tearing of the pancreas.

After tearing of the pancreas, a secretory inhibition of the pancreas occurred, which was resistant even to powerful stimulation in the immediate postoperative period and during the first postoperative days. This gave the wound of the pancreas a chance to be sealed firmly by omentum.

The presence of devitalized tissue in the peritoneal cavity did not seem to affect adversely the healing of a pancreatic injury. However, the formation of inflammatory masses around the necrotic tissue seemed much more pronounced in the cases with simultaneous injuries to the pancreas than in the control cases. Clinical suggestions are drawn from the results of these experiments.

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STUDIES ON VAGOTOMY IN THE TREATMENT OF PEPTIC ULCER

V. Clinical and Physiologic Results Three to Four Years After Surgery

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THE use of complete vagotomy in the surgical treatment of peptic ulcer was first advocated by Dragstedt and Owens in 1943 (4). Previous to this time, partial vagotomy had been employed by numerous surgeons but was abandoned as ineffective. The voluminous literature on this subject is presented in recent review articles (1, 12, 23). Our studies on vagotomy were started in June, 1946, and the results of the first phase of this work have been previously published (10, 19, 20, 22).

In addition to what has been established in the literature, there are two fundamental questions which remain to be answered before an accurate final evaluation of vagus section can be attempted. Clinically, it must be ascertained what proportion of patients operated upon continue to have unpleasant symptoms or have a recurrence of ulcer symptoms after a number of years. From a physiologic viewpoint it is necessary to determine if the marked decrease in gastric secretory capacity and motility found in the early postoperative period (19), are permanent following complete vagotomy. There have been no reports in the literature as yet concerning the physiologic effect of vagotomy 3 to 4 years after surgery.

At present, patients who have had vagus section are designated as incomplete or complete vagotomy according to the results of the insulin test. Data will be presented showing that a functional division is more accurate on the basis of normal vagus function, partial vagus function, and no vagus function.

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Fifty peptic ulcer patients have been followed clinically after vagotomy. Forty-four of these have been subjected to physiologic studies and 25 patients were restudied 3 to 4 years following vagotomy. This is a relatively small group for the evaluation of clinical results, but a relatively large group to have had careful and repeated physiologic studies. The clinical results, therefore, will be presented in summary form, whereas the physiologic studies will be presented in detail.

SUMMARY OF CLINICAL DATA

Vagotomy was performed upon 50 peptic ulcer patients. The patients were selected for surgery on the basis of severe ulcer diathesis as evidenced by a long complicated history of ulcer disease (10). Approximately 50 per cent of the patients had had two or more serious ulcer complications before vagotomy. Thirty-three of the patients had duodenal ulcers and 17 had anastomotic ulcers.

There were 3 deaths. One patient died on the eighth postoperative day from lower nephron nephrosis due to a blood transfusion reaction. One died 3 months postoperatively from bronchopneumonia and chronic alcoholism. The third died 16 months following surgery, from a coronary occlusion. Postmortem examination in each of these patients showed, in addition to the above, a healing duodenal ulcer in the first patient and healed duodenal ulcer in the 2 others. The follow-up was unsatisfactory in 7 patients, leaving a total of 40 who were followed 28 to 50 months. Twenty-eight were followed for more than 36 months.

The clinical results in these 40 patients are as follows: good, 26; fair, 10; poor, 4. The patients with good results had no ulcer symptoms and no lasting complications from vagus section. The patients with fair results had

TABLE I.—NUMBER OF GASTRIC FUNCTION TESTS PERFORMED BEFORE AND AFTER VAGOTOMY

	Night secretion	Basal secretion	Insulin	Histamine	Caffeine
Preoperative and early postoperative	158	188	76	44	44
	(22)	(22)	(38)	(22)	(22)
Late postoperative	0	73	26	26	14
		(25)	(25)	(25)	(14)
Total	158	261	102	70	58

Number of patients in parentheses.

vague complaints or possible mild ulcer recurrences, but were definitely improved. One of the patients classified as a poor result has had frequent, mild ulcer recurrences, but the patient states that his ulcer is more easily managed than before surgery. The 3 remaining patients with poor results are the only ones in this series upon whom reoperation was performed for gastroduodenal disease. Each of these patients was a chronic alcoholic. In 2 of the patients, after vagotomy, achlorhydria was present and no evidence of vagus function was found after carefully repeated tests. Reoperation was advised in each case because of massive, recurrent upper intestinal hemorrhage. There was no evidence at surgery of active peptic ulceration in any of these cases. All 3, however, had a marked polypoid, hypertrophic hemorrhagic gastritis.

Postoperative vagotomy complications such as poor gastric emptying, diarrhea, and cardiospasm were transient and occurred infrequently when proper management was employed following surgery (5, 10, 21).

Discussion. This group of patients was selected on the basis of frequent severe ulcer complications before vagotomy. It is emphasized that there have been no severe ulcer complications following vagus section in this series. One case of definite ulcer recurrence is mentioned. There are 6 other cases of possible ulcer recurrence (listed in results under "fair") in whom mild transient, ulcer-like symptoms have occurred once or twice over a period of 3 to 4 years. The ultimate results in these patients is as yet unknown.

Three of the 4 patients with poor results were chronic alcoholics. Four of the patients with fair results were chronic or intermittent alcoholics. It appears that alcohol may have a more damaging effect upon the stomach following vagotomy. Although we have not suggested any routine ulcer management to our patients following vagotomy, we strongly advise against the use of alcoholic beverages.

PHYSIOLOGIC ASPECT

Method and tests. Preoperative and postoperative physiologic studies of the stomach were made a few weeks before and after vagotomy and similar late studies were repeated 3 to 4 years later. A total of 44 patients was tested, and of this group 25 patients received tests during the late period, 3 to 4 years after vagus section. Four patients with previous gastric resection were excluded, leaving 21 comparable patients with duodenal ulcer who were subjected to complete secretory studies, including preoperative, early postoperative, and late postoperative gastric function tests. The number and type of secretory tests performed in this study are shown in Table I. In addition to these studies, 27 patients were subjected to gastric motility determinations before and after surgery which totaled approximately 10 hours for each patient. During the late postoperative period, gastric motility was determined for approximately 5 hours in each of 25 patients.

The reliability of the individual test was established by the fact that the preoperative tests showed consistent results without need for repetition. In addition, all of the tests were performed by one investigator.

1. The insulin test. The principles and method of using both secretory and motor gastric response to insulin hypoglycemia as a test of vagus function have been described in detail (20). After a 12 hour fast, a Levin tube was passed into the stomach for collection of the gastric secretion. After the gastric residuum was removed, a second Levin tube, with a rubber balloon attached, was passed into the stomach. The balloon capacity was about 200 cubic centimeters but in order to avoid mechanical stimulation, only 10 to 15 cubic centimeters of air were used during the test. The balloon was placed in the cardiac end of the stomach and its position confirmed by inflation and gentle withdrawal of the attached

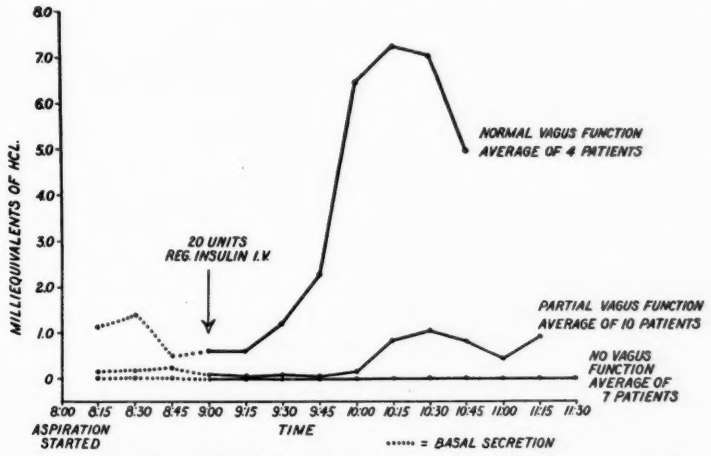

Fig. 1. The effect of insulin hypoglycemia on gastric acidity 3 to 4 years after vagotomy.

tube until there was a slight tug as the balloon reached the cardiac sphincter of the stomach. By this means, it was assured that the balloon was not in the pylorus where it might cause mechanical stimulation. The balloon was then only partially inflated and connected to a water manometer. Continuous recordings of gastric motility were made on a slowly moving kymograph. The first tube was advanced about 2 inches beyond the balloon in order to prevent aspiration from interfering with the balloon. A control period of at least 1 hour of basal secretion and motility was observed. The gastric secretions were aspirated continuously through the first tube and collected at 15 minute intervals for titration. Then 15 to 20 units of regular insulin were injected intravenously and the effect on gastric motility and acidity noted for 2 or 2½ hours.

2. *Basal secretion.* Basal secretion is the spontaneous secretion of the unstimulated stomach (2, 9). It was determined in the morning after a 12 hour period of fasting. A Levin tube was passed into the stomach, the gastric residuum removed, the stomach kept empty by means of continuous syringe aspiration, and the specimens of gastric secretion collected at 15 minute intervals for titration. The basal secretion was determined for at least a 1 hour period before any other test of gastric function was started. In some instances, basal secretion and spontaneous gastric motility were determined simultaneously.

Determination of the 12 hour night secretion was made in the preoperative and postoperative period, but was omitted during the late studies for two reasons. Many of the patients refused to enter the hospital for the late studies, but were willing to be tested as outpatients. In addition, our previous studies demonstrated that the basal secretion is a simple accurate method of determining the interdigestive secretory activity of the stomach and that the effect of vagotomy on the night secretion and basal secretion is similar (19).

3. *The histamine test.* Following a period of at least 1 hour of basal secretion, an intramuscular injection of 1.0 milligram of histamine diphosphate was given. Aspiration of the stomach was continued for 1 hour as during the basal period and 15 minute samples were collected for titration.

4. *The caffeine test.* The method employed was that of Roth, Ivy, and Atkinson (16). After the determination of basal secretion, 0.5 gram of caffeine with sodium benzoate in 200 cubic centimeters of water was given through the Levin tube. Aspiration of the gastric contents was resumed one-half hour later and continued for 2 hours.

Results of tests. The insulin test. Results of the insulin test 3 to 4 years after vagus section

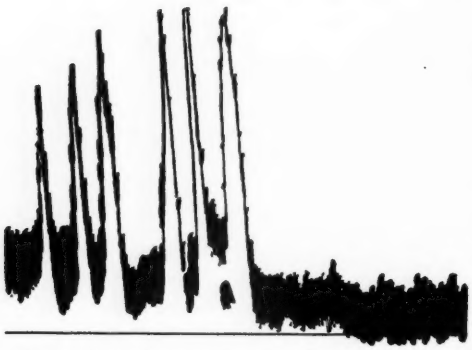

Fig. 2. A 15 minute portion of a 5 hour gastric motility tracing, illustrating the normal gastric hunger contractions of patients with normal vagus function 3 to 4 years after vagotomy.

demonstrate 3 types of response on the basis of vagus function (Fig. 1). The first type, designated as normal vagus function, is characterized by a secretory response similar to that obtained preoperatively in all of the duodenal ulcer patients. The second is designated as partial vagus function on the basis of a slight, but definite acid response to insulin hypoglycemia. The third, designated as no vagus function, is characterized by no acid response to insulin hypoglycemia.

Twenty-one comparable patients were studied. Four patients with normal vagus function had an average secretory rate of 27.7 milliequivalents per hour with a range of 17.4 to 36.3. The average secretory rate of 10 patients with partial vagus function was 4.0 milliequivalents per hour with a range of 0.0 to 7.5. There was *no* secretory response to insulin hypoglycemia in 7 patients with no vagus function.

Gastric motility was determined in each of the 25 patients studied. Typical gastric hunger contractions, similar to those present in all of the patients before vagus section, were demonstrated in each of the 4 patients with normal vagus function (Fig. 2). The 10 patients with partial vagus function had irregular low gastric contractions (Figs. 3 and 4). In some instances spontaneous motility was present during the fasting period, whereas occasionally, motor activity was not present until several hours after the insulin hypoglycemia. The 7 patients with no vagus function had occasional tonus rhythm, but no hunger contractions (Figs. 5 and 6). The gastric motility pattern of each group conformed accurately with the functional divisions as determined by the acid response.

Fig. 3. A 15 minute portion of a 5 hour gastric motility tracing, illustrating the typical irregular low gastric contractions in patients with partial vagus function 3 to 4 years after vagotomy.

This functional division of the patients into 3 groups on the basis of the insulin test is utilized in the presentation of the results of the histamine and caffeine tests and the basal secretion studies.

Basal secretion. Sixty-two determinations of basal secretion were conducted on 20 comparable patients. Figure 7 represents the average basal secretion 3 to 4 years after vagotomy grouped on the basis of vagus function. The average basal secretion of 9 determinations in 4 patients with normal vagus function was 4.0 milliequivalents per hour. (Before vagotomy the average basal secretion of 56 determinations in 22 patients was 3.7 milliequivalents per hour.) The average basal secretion of 30 determinations in 9 patients with partial vagus function was 1.3 milliequivalents per hour. There was *no basal secretion* of free hydrochloric acid in 23 determinations in 7 patients with no vagus function.

The histamine test. Nineteen patients were tested with histamine 3 to 4 years after vagotomy. The results of these tests have been grouped according to vagus function in Figure 8. The average secretory response of 3 patients with normal vagus function was 9.8 milliequivalents per hour. (The average preoperative response of 11 patients was 12.7 milliequivalents per hour.) The average response of 9 patients with partial vagus function was 4.7 milliequivalents per hour. The average response of 7 patients with no vagus function was 1.9 milliequivalents per hour.

Fig. 4. A 15 minute portion of a 5 hour gastric motility tracing, illustrating the typical irregular low gastric contractions in patients with partial vagus function 3 to 4 years after vagotomy.

Fig. 5. A 15 minute portion of a 5 hour gastric motility tracing, illustrating the occasional tonus rhythm but no hunger contractions in patients with no vagus function 3 to 4 years after vagotomy.

The caffeine test. Fourteen patients were tested with caffeine 3 to 4 years after vagotomy. The results of these tests have been grouped according to vagus function in Figure 9. The secretory response of 1 patient with normal vagus function was 16.9 milliequivalents per hour. (This was much greater than the average preoperative response.) The average response of 8 patients with partial vagus function was 2.3 milliequivalents per hour. The average secretory response of 5 patients with no vagus function was 0.7 milliequivalents per hour.

Discussion. Two questions arise concerning the division of patients into groups on the basis of vagus function. First, is this subdivision valid, and second, what is its purpose? The reason for this functional grouping will be discussed in detail. The initial division of the patients into the 3 functional groups was determined by the acid response of the stomach to insulin hypoglycemia, which is the most accurate method at present of determining vagus function. Thus each patient was placed in a group as demonstrating either normal vagus function, partial vagus function or no vagus function. The term normal vagus function refers to the usual vagus function observed in peptic ulcer patients and does not necessarily apply to patients without ulcer diathesis. The patients with partial vagus function have a decreased gastric secretory capacity, and only slight evidence of vagus function. We have been unable to obtain any evidence of vagal activity in the group of patients with no vagus function.

It could be argued that the group which has been designated as having partial vagus function are simply incomplete vagotomies in persons with inherently low secretory capacity. However, this possibility is ruled out by the fact that the preoperative secretory studies in this group showed in general the same high level of response as the other two groups.

It has been previously shown that the secretory capacity of the stomach, whether during periods of spontaneous or stimulated secretion, is in some manner dependent upon vagus function (6, 13, 18, 19). The validity of the functional vagus grouping is substantiated by the fact that results of the secretory tests (basal secretion, histamine, and caffeine) conformed accurately with the grouping as determined by the acid response to insulin hypoglycemia. This relationship proved to be so reliable that the appearance of any free acid during a period of basal or night secretion became an indication that vagus function was present.

In addition to this excellent correlation of the secretory data, the gastric motility pattern

Fig. 6. A 15 minute portion of a 5 hour gastric motility tracing, illustrating the occasional tonus rhythm but no hunger contractions in patients with no vagus function 3 to 4 years after vagotomy.

of each group conformed accurately with the functional divisions as determined by the acid response to insulin hypoglycemia. This relationship proved to be so dependable that the presence of "vagal type motility," as shown in Figures 2, 3, and 4, can be used as evidence of the presence of vagal activity. Thus when gastric resection has been previously performed, and the acid response may be unreliable, the motor activity of the fundus of the stomach can be used as a reliable index of vagus function.

The patients were subdivided into the 3 functional groups in order that accurate data could be obtained concerning possible return of gastric function following vagotomy. In 1929, Hartzell reported a reduction in gastric acidity in the dog, 1 to 5 months after vagotomy (7). Vanzant (24, 25) restudied the problem in dogs and reported a gradual return of function first noted about 6 months after vagus section. Moore and Ruffin have recently indicated an early return of function following vagotomy in man, although Postlethwait and Deaton noted no evidence of a decided return to preoperative secretory or motor levels in 7 dogs 1 and 2 years following vagotomy (3, 15). Walters recently reported that a return to preoperative acid levels occurred in 23.7 per cent of 85 cases 1 to 4 years after vagotomy as measured by an Ewald test. Night secretion studies were not made and histamine tests not reported. Thus there has been no agreement as to whether return of gastric function takes place following vagotomy.

Having divided the patients into 3 functional groups, a comparison can be made for each group between the preoperative and early and late postoperative secretory studies in order to evaluate possible return of gastric function. The preoperative studies were conducted a week or two before vagotomy, and the early postoperative studies in the second week following vagus section (19). The late postoperative studies were done 3 to 4 years later. Patients were used for this comparison only when data were available from each of the three periods of study, thus the same number and group of patients were used for each individual comparison.

Normal vagus function was demonstrated in the late period in 4 patients. There was no significant difference between the preoperative, early postoperative and late studies in this group of patients.

Partial vagus function was noted in the late postoperative period in 10 patients. Complete data for the 3 periods of study were available in 6 of these patients concerning the insulin test and basal secretion. Similar data were available for the histamine test in 3 patients and for the caffeine test in 4 patients (Table II). The secretory rate of all of the tests for each patient was markedly decreased during the early and late postoperative periods as compared to the preoperative response. It appears from Table II that there is a greater response to insulin during the late postoperative period than during the early postoperative period, and thus, that there is a late return of gastric function. This may be true, but no conclusions can be made because the technique of performing the insulin test was changed in a manner which may have produced this apparent increase in the insulin response. The early insulin tests were continued for 1 to 1½ hours following the injecting of insulin, at which time 5 of the 6 patients showed a slight rise in free acid. Moore noted this phenomenon and stated that in some cases the acid response to insulin hypoglycemia may be delayed. During the late studies a negative insulin test was not considered adequate unless it was continued for at least 2½ hours after the injection of insulin. (In addition, of course, adequate hypoglycemia is essential.) The secretory rate was calculated in each instance for the hour of greatest secretion following the hypoglycemia. Thus, this delayed response was probably missed in the early postoperative studies in this group of patients. The delayed response appears to be characteristic of patients with partial vagus function as shown in Figure 1.

Analysis of the individual early postoperative secretory records of these patients with late partial vagus function show that partial function was present early in 5 patients as demonstrated either by spontaneous free acid secretion, or by a slight acid response to hypoglycemia.

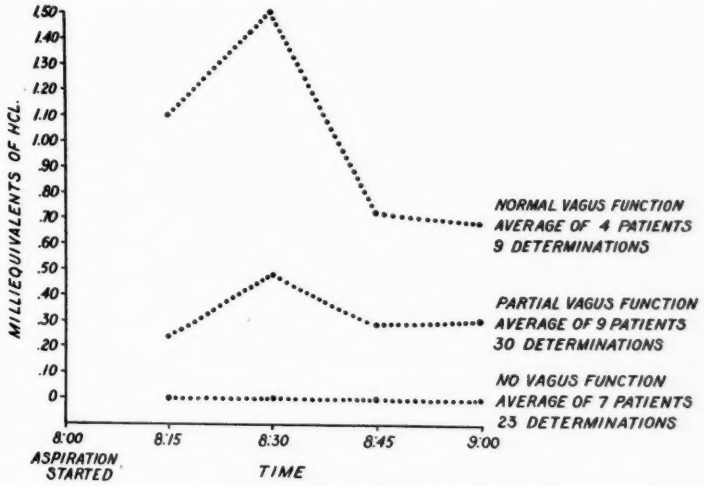

Fig. 7. Basal secretion 3 to 4 years after vagotomy.

The average basal secretory rate during the late postoperative period for these 6 patients with partial vagus function was 1.67 milliequivalents per hour and each patient secreted free hydrochloric acid under basal conditions. The average basal secretory rate for these same patients during the early postoperative period was 0.67 milliequivalent per hour; at this time 2 of these patients had no basal free hydrochloric acid. Statistical analysis of the basal secretion data shows that this increase is significant (probability, 0.04). On the other hand, the slight increase in response to histamine and the slight decrease in response to caffeine in the late period are not statistically significant.

It can be stated, therefore, that in patients with *partial vagus function* there is a slight increase in basal secretion 3 to 4 years after surgery. It should be emphasized, however, that in spite of this increase over the early postoperative result, there is still a 66.2 per cent decrease when compared to the preoperative secretory rate, and that there remains a 70 to 80 per cent decreased response to histamine and caffeine in the late period as compared to the preoperative response.

No vagus function was demonstrated during the late period in 7 patients. Complete data for the three periods of study were available in 5 of these patients concerning the insulin test and basal secretion. Similar data were available for the histamine test in 5 patients and for the caffeine test in 2 patients (Table III). Comparison of the early and late postoperative secretory rate for insulin, basal secretion and histamine, show that there has been an apparent small *decrease* in the secretory rate. In addition, 3 of the 7 patients secreted small amounts of free acid during basal testing in the early postoperative period whereas none of them did during the late period. These 3 patients also had a slight response to hypoglycemia, and thus showed partial vagus function during the early period and no vagus function late. This decrease may be explained by the possibility that a small number of functional vagus fibers remained intact following surgery, and later lost their function due to the trauma of surgery or to scar formation. At least one of these patients, however, had a moderate gastritis, which could be present in some of the others and explain this decreased secretory rate. There is an insignificant increase in the late secretory rate following caffeine stimulation.

The finding to be emphasized in this group of patients is that there is no evidence of return of secretory vagal function 3 to 4 years following vagus section. In addition, there is

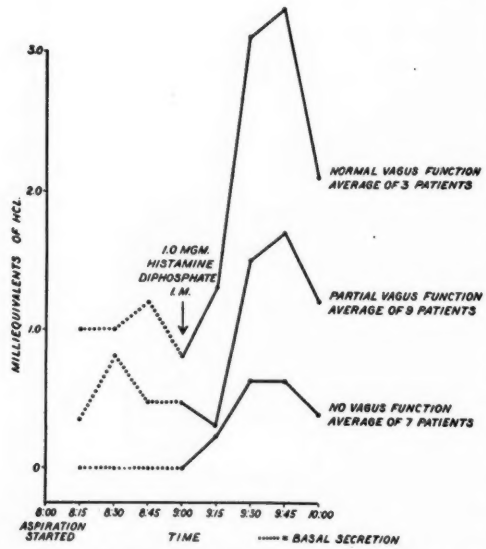

Fig. 8. The effect of histamine on gastric acidity 3 to 4 years after vagotomy.

TABLE II.—COMPARISON OF PREOPERATIVE AND EARLY AND LATE POSTOPERATIVE SECRETORY RATES IN A GROUP OF PATIENTS SHOWING PARTIAL VAGUS FUNCTION 3 TO 4 YEARS AFTER VAGOTOMY

Secretory test	Number of patients	Average secretory rate of HCl mEq./hr.	Per cent decrease from preoperative value
Insulin			
Preoperative	6	14.3	—
Early postoperative	6	1.87	86.9
Late postoperative	6	5.05	64.6
Basal secretion—			
Preoperative	6*	4.94	—
Early postoperative	6†	0.67	86.4
Late postoperative	6‡	1.67	66.2
Histamine			
Preoperative	3	17.0	—
Early postoperative	3	3.4	80.0
Late postoperative	3	4.5	73.5
Caffeine			
Preoperative	4	12.1	—
Early postoperative	4	3.5	71.0
Late postoperative	4	2.4	80.1

*15 determinations
†23 determinations
‡21 determinations

no evidence of return of vagal motor activity in this group of patients.

Some patients with so called "incomplete vagotomy" have had good clinical results. It is emphasized that many of these patients have only *partial vagus function* with a marked persistent depression of gastric secretory and motor activity. Fourteen of our patients had a positive insulin response; however, 10 of these exhibited only partial vagus function. Weinberg recently reported quantitative results of the postoperative insulin test in 350 patients. Seventy-one of 90 patients with a positive reaction, but no ulcer symptoms, had a free acid response of 20 clinical units or less, and are probably patients with partial vagus function. Only 1.5 per cent of the patients with a negative response had persistence or recurrence of ulcer symptoms whereas 5 of 8 failures occurred in patients with a high acid response to insulin. In our series, 4 of 6 patients with normal vagus function have had definite or suspected ulcer recurrences. Three of 17 patients with partial vagus function have had short periods of vague abdominal distress, but no definite ulcer recurrences. None of 10 patients with no vagus function have had ulcer recurrences, but 2 had massive gastric bleeding due to gastritis following chronic alcoholic consumption. (These figures include some patients in whom the follow-up is less than 3 years.) Thus there has been good correlation between the clinical results and the insulin test in our patients and those reported by Weinberg. Dragstedt's findings are similar (5). Several papers (26, 28) have indicated a lack of correlation between the results of the insulin test and the clinical outcome. This is probably due to the previous failure to recognize the existence of the intermediate group of patients with partial vagus function in whom there is a markedly decreased secretory capacity.

Physiologically it has been considered in the past that so called "incomplete vagotomy" has little if any effect upon gastric function. Although this is apparently true in certain instances, such as patients who have normal vagus function following an attempted vagotomy, it is now clear that there is a definite group of patients with partial vagotomy who

demonstrate a marked, long-lasting decrease in gastric secretory and motor activity. There are two possible explanations for the physiologic mechanism of partial vagus function. First, the small number of intact vagal fibers remaining after surgery in some cases may supply vagal innervation to only a small portion of the stomach. Second, it is possible that *all* vagus fibers have not been sectioned in any of the cases, and that in those patients with a high initial secretory capacity, vagus function, although slight, can be demonstrated. In other patients, in whom the initial secretory capacity was not as great, no vagus function can be shown although a few fibers may remain. It appears from Tables II and III that patients who demonstrated partial vagus function following surgery had a higher preoperative secretory rate for each test than the patients in whom subsequent tests showed no vagus function. Analysis of the individual results, however, shows wide overlap and does not support this possibility.

The necessity for dividing the patients into 3 functional groups first became apparent when the results in the patients now grouped as partial vagus function were compared to the early postoperative results, at which time some of them were designated as "complete vagotomy." It first appeared that an obvious return of function was present in these patients. However, when the early postoperative results were divided into the functional groups, it became clear that the group with partial vagus function had a greater early postoperative secretory rate than the group with no vagus function.

On the basis of these findings, several methods can be utilized for determining the presence of vagus function. The acid response of the stomach to hypoglycemia remains the preferred method, and even a small free acid response is significant. The appearance of a spontaneous secretion of free hydrochloric acid during periods of night or basal secretion indicates presence of vagus function. Also, the appearance of vagal gastric motility, either spontaneous or after hypoglycemia, is a third index of the presence of vagus function.

A final physiologic consideration remains concerning the relationship of vagotomy to

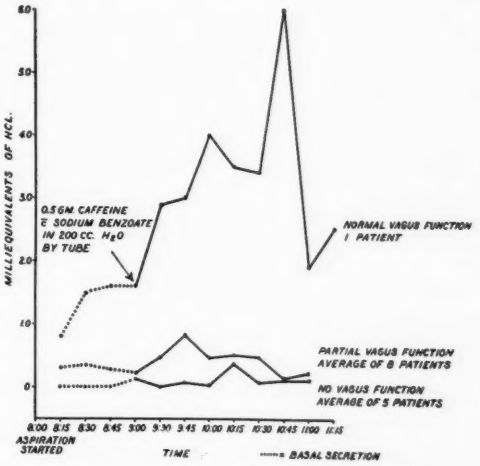

Fig. 9. The effect of caffeine on gastric acidity 3 to 4 years after vagotomy.

the mechanisms of gastric secretion and motility. Ivy in 1925 proposed that the secretory mechanism of the stomach should be divided

TABLE III.—COMPARISON OF PREOPERATIVE AND EARLY AND LATE POSTOPERATIVE SECRETORY RATES IN A GROUP OF PATIENTS SHOWING NO VAGUS FUNCTION 3 TO 4 YEARS AFTER VAGOTOMY

Secretory test	Number of patients	Average secretory rate of HCl mEq./hr.	Per cent decrease from preoperative value
Insulin			
Preoperative	5	11.6	—
Early postoperative	5	0.1	99.1
Late postoperative	7	0.0	100.0
Basal secretion—			
Preoperative	5*	3.15	—
Early postoperative	5†	0.3	90.5
Late postoperative	7‡	0.0	100.0
Histamine			
Preoperative	5	13.4	—
Early postoperative	5	5.0	62.8
Late postoperative	5	2.4	82.1
Caffeine			
Preoperative	2	10.9	—
Early postoperative	2	0.4	96.3
Late postoperative	2	1.0	90.8

*12 determinations
†13 determinations
‡23 determinations

into two periods, the interdigestive and the digestive, and that the latter should be divided into 3 phases, the cephalic, the gastric, and the intestinal. Gastric secretion during the interdigestive period takes place in the absence of food stimuli. Secretion during the digestive period is in response to food stimuli, acting through the sensorium, the stomach, or the intestine. In addition, emotional factors may stimulate or depress the gastric activity during any period, and they are classified as emotogenic.

From the data presented in Table III, it can be seen that when no vagus function can be demonstrated by the insulin test, there is no basal secretion of free hydrochloric acid. Thus the interdigestive period of gastric secretion, as measured by basal secretion, is decreased 100 per cent. It has been previously shown that the cephalic phase of the digestive period is abolished by vagus section (14). There is a profound and lasting depression of the gastric phase of the digestive period in patients with no vagus function following surgery, as shown by an 82 per cent decrease in the response to histamine and a 90 per cent decrease in the response to caffeine 3 to 4 years later. Because there is a decreased secretory response of the stomach to the strongest known stimulus, histamine, it is likely that the response of the stomach to stimuli acting during the intestinal phase of the digestive period is also decreased. The effect of emotogenic factors upon the stomach is abolished by vagotomy (29). In addition, there is a persistent absence of gastric hunger contractions. It is emphasized that in patients with no vagus function, there is a marked lasting depression of the *entire mechanism* of gastric secretion and motility. There is a similar although less marked decrease of gastric secretory and motor activity in patients with partial vagus function. The notion that vagotomy interferes only with the cephalic (reflex or psychic) phase of secretion is obviously inadequate. The effect of vagotomy in reducing the response to direct stimulants such as histamine and gastrin has been attributed to the loss of the sensitizing of potentiating action of acetylcholine which results from the removal of cholinergic impulses (6).

SUMMARY AND CONCLUSIONS

Fifty peptic ulcer patients have been followed clinically after vagotomy. Forty-four of these patients have been subjected to physiologic studies and 25 patients were subjected to repeat studies conducted 3 to 4 years following vagotomy.

Results of physiologic studies demonstrate 3 types of response on the basis of vagus function, which have been termed: (1) normal vagus function, (2) partial vagus function, and (3) no vagus function. No significant change in the gastric secretory or motor activity is present in patients with normal vagus function following surgery. A marked reduction in the motility and in both spontaneous and stimulated acid secretion of the stomach is demonstrated in patients with partial vagus function and those with no vagus function 3 to 4 years after vagotomy. This reduction is greatest in patients with no vagus function. No evidence of any return of gastric activity is present in this group. A slight return of acidity is found in patients with partial vagus function as determined by the basal secretion; however, in spite of this, there is still a marked decrease in comparison with the preoperative level.

The previous failure to recognize the existence of the intermediate group (partial vagus function) probably accounts for reports concerning the lack of correlation between the physiologic and therapeutic effects of vagotomy. The response of patients with partial vagus function to insulin hypoglycemia is characteristically delayed, and may be missed unless the test is continued for at least 2½ hours after the injection of insulin. The appearance of a spontaneous secretion of free hydrochloric acid during periods of night or basal secretion following vagotomy indicates the presence of vagus function. In addition, the appearance of vagal type motility, either spontaneous or subsequent to hypoglycemia, constitutes an index of the presence of vagus function.

The marked decrease in gastric motor and secretory activity following vagotomy in patients with partial vagus function and no vagus function is persistent 3 to 4 years after surgery.

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MAJOR SURGERY IN HODGKIN'S DISEASE

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THE indications for surgery in patients with Hodgkin's disease and the surgical risk in these patients has, to our knowledge, never been fully presented. A summary of the surgery and its effects upon the Ohio State University Medical Center patients proved to have Hodgkin's disease is consequently submitted.

Between January, 1940 and July, 1950 approximately 400 cases of Hodgkin's disease were seen at the Ohio State University Medical Center. Two hundred and forty of these patients were hospitalized for study and treatment. Thirty-one, or 12.9 per cent, were subjected to one or more major surgical procedures. In only 1 patient was a direct surgical attack made with the hope of cure. Surgery was used in the remaining 30 cases in the treatment of complications arising from the disease or unrelated conditions occurring during its course. These latter cases have been studied to determine the indications for surgery, the effect of surgical intervention upon the course of Hodgkin's disease, and the influence of Hodgkin's disease upon the ability of the patient to withstand surgery.

The average age of 18 males and 13 white females requiring surgery was 38 years. In all cases reviewed, the diagnosis of Hodgkin's disease was established by a biopsy and frequently confirmed by one or more subsequent biopsies. Thirty-five operations were performed upon the 31 patients.

PRESENTATION AND DISCUSSION OF CASES

The literature on Hodgkin's disease contains little regarding definitive surgery in the treatment of this disease process and practically nothing regarding major surgery for conditions either directly or indirectly related to the disease (3). The 35 operative procedures in this series include: 1 radical neck dissection, 6 thoracotomies, 23 laparotomies, 1 mastectomy, 1 drainage of a perirenal abscess, and 3 laminectomies.

The indications for surgery in Hodgkin's disease can be classified into four categories:

1. Surgery for complications directly or indirectly related to the Hodgkin's disease—hypersplenism, pressure symptoms (spinal cord, trachea, etc.), localized large growths causing pain or limiting adequate x-ray or chemotherapy.

2. Surgery for diagnosis—peripheral node biopsy, thoracotomy, laparotomy.

3. Surgery for conditions unrelated to Hodgkin's disease.

4. Surgery for definitive treatment of the Hodgkin's disease.

These indications are presented in the order of their frequency and will be discussed with illustrative cases.

SURGERY FOR COMPLICATIONS DIRECTLY OR INDIRECTLY RELATED TO HODGKIN'S DISEASE

Complications arising during the medical management of patients with Hodgkin's disease presented the most frequent indication for surgical intervention. In our series the most frequent complication has been hypersplenism; and our results establish conclusively the value of splenectomy in these patients. The mechanism of secondary hypersplenism has not been explained satisfactorily but has been observed in many disease processes which may involve the spleen (2). Involvement of the spleen by Hodgkin's disease is common (3). Hodgkin's disease of the spleen may be accompanied by a hypersequestration and destruction of any one or all of the formed elements of the peripheral blood. The bone marrow picture at that time represents a hyperplasia of megakaryocytes if the peripheral platelet count is diminished, hyperplasia of the erythroid elements in the event of secondary hemolytic anemia, hyperplasia of the myeloid elements with a peripheral neutropenia, or a marrow hyperplasia involving all cellular

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elements accompanying a peripheral pancytopenia.

Eleven splenectomies were performed for hypersplenism. In 4 of these cases the preoperative impression was that the associated thrombocytopenia or neutropenia was due to primary hypersplenism. The fact that the hypersplenism was secondary to Hodgkin's disease in these 4 cases was first established at the time of splenectomy. The diagnosis of Hodgkin's disease was established before the development of hypersplenism in the 7 other cases. All of these cases had splenomegaly concomitant with the hypersplenism. Following splenectomy the hematologic pattern returned to normal or was greatly improved in all but 2 of the 11 cases. The operative and postoperative courses were uneventful. These patients survived from 1 to 28 months following splenectomy thus showing prolongation of life by splenectomy in the patient with Hodgkin's disease who develops secondary hypersplenism.

An evaluation made of the degree of activity and the rate of progress of the primary disease before surgery in each case and a comparison of the length of therapy-induced remissions before and after operation suggest that the surgical procedure did not accelerate the course of the Hodgkin's disease in any of these individuals.

The following cases illustrate the response of secondary thrombocytopenia, neutropenia, pancytopenia to splenectomy in Hodgkin's disease:

D. L. (Case 8) developed jaundice, reticulocytosis, and a marked anemia 2 years after the onset of Hodgkin's disease. Bone marrow studies demonstrated a significant erythroid hyperplasia. A splenectomy was performed without operative or postoperative complications and the red blood cell count returned to normal. The Hodgkin's disease continued to follow a chronic course. Fourteen months later a thrombocytopenia developed and a megakaryocytic hyperplasia was found in the bone marrow. Thorotrast was administered intravenously and a subsequent x-ray film of the abdomen demonstrated localization of that substance in an accessory spleen. Following the removal of the accessory spleen, the platelet count returned to normal and the hemorrhagic manifestations stopped. The patient continued to pursue a chronic course and expired of generalized Hodgkin's disease 27 months after the second laparotomy.

A. M. (Case 12) developed thrombocytopenic purpura 16 months after the onset of Hodgkin's disease and after the institution of radiation therapy. An uncomplicated splenectomy was performed and the thrombocytopenia corrected. No apparent alteration in the course of Hodgkin's was observed, and the patient expired 8 months later.

H. O. (Case 7) developed neutropenia with bone marrow myeloid hyperplasia 17 months after the onset of a rapidly progressing type of Hodgkin's disease. Response to specific therapy for the Hodgkin's disease had been brief and incomplete throughout the course of the disease. A splenectomy was performed, followed by a slow postoperative recovery. The white cell count, however, rose during the week following surgery to normal levels and subsequently a leukocytosis developed.

J. M. (Case 11) was found to have a pancytopenia during the advanced phase of Hodgkin's disease. A splenectomy was followed by an increase in circulating red blood cells and platelets to normal levels.

G. B. and R. Y. (Cases 6 and 9) developed hemolytic anemia during the course of Hodgkin's disease. Splenectomy produced no hematologic improvement in either case.

Compression symptoms offer the second most frequent indication for surgery (4 cases). Spinal cord compression from epidural Hodgkin's disease may become a surgical emergency. Three laminectomies were performed for epidural Hodgkin's disease of the spinal cord. Immediate neurological improvement was noted in 1 patient operated upon early, delayed improvement in 1 patient operated upon 1 week after the development of paralysis, and slight improvement but no progression of symptoms in a case in which neurologic manifestations had been present longer.

Our observations are in agreement with Smith and Stenstrom who state that impending cord compression may be relieved by x-ray therapy. When evidence of cord compression exists, however, immediate laminectomy and excision of the Hodgkin's tissue is indicated before roentgen radiation is given. X-ray therapy in doses required for benefit is frequently followed by edema of the radiated tissues. If a laminectomy has not been done to allow room for expansion, this may aggravate the cord compression or produce irreversible damage. The results of surgery may be dramatic and, if followed by x-ray therapy, may be prolonged. The same principles apply for tracheal, gastrointestinal, and genitourinary tract compression.

A third complication arising as a result of Hodgkin's disease is pain due to extensive growth of Hodgkin's tissue. Removal of large masses of accessible Hodgkin's tissue may produce symptomatic improvement in conjunction with adequate roentgen radiation. Primary Hodgkin's disease of the breast is rare, and secondary Hodgkin's disease of the breast rarely responds to surgical treatment (1). In 1 case in this series there was massive involvement of the breast. Mastectomy was performed to remove the large painful mass and also as an adjunct to x-ray therapy. Improvement following surgery was slight and temporary, the patient expiring from generalized Hodgkin's disease 5 months later. The slow downhill course did not appear to be either accelerated or retarded by surgery.

SURGERY FOR DIAGNOSIS

Peripheral lymph node biopsy is a well accepted surgical procedure and requires no discussion in this presentation. While in most patients suspected of having Hodgkin's disease the diagnosis can be substantiated by peripheral lymph node biopsy, occasionally patients are seen who have no evidence of lymph node enlargement, or, if adenopathy is present pathologic examination fails to establish the diagnosis. When there are findings suggestive of involvement of the chest or abdomen, the question arises as to the advisability of exploratory thoracotomy or laparotomy. Major diagnostic surgical procedures have not been so widely used, perhaps due to the belief that these procedures are rarely warranted in patients considered to be "poor surgical risks." The information obtained by surgery, however, may be of utmost importance in establishing a diagnosis and in guiding future therapy.

Five patients in this series had exploratory thoracotomies performed. In 2 of these patients, mediastinal tumors causing tracheal compression were removed at the time of thoracotomy to relieve these symptoms as well as to establish the diagnosis. In 2 other patients without pressure symptoms, a lung biopsy and a biopsy of a mediastinal mass established the pathological diagnosis for the first time. Four patients withstood the procedures well and survived 11 to 40 months postoperatively. The fifth patient developed cardiac arrest during the operative procedure; and despite successful manual resuscitation, this patient expired in shock shortly following completion of the thoracotomy.

Six laparotomies were performed for the purpose of establishing the diagnosis of Hodgkin's disease. In these patients the physical findings were those of a chronic debilitating disease with no peripheral adenopathy. In 4 of these 6 cases there was a palpable abdominal mass. At laparotomy, biopsy of liver, spleen, and abdominal nodes was performed. The diagnosis was accurately established by laparotomy; there were no operative complications, and in 4 of these cases the postoperative course was uneventful. The 2 cases in which postoperative complications developed are presented in more detail.

J. F. (Case 21) was admitted to the hospital with fever, icterus, and ascites of 2 months' duration. Two years prior to this admission a diagnosis of Hodgkin's disease had been made by cervical lymph node biopsy and the patient had responded to small doses of x-ray therapy until the development of pneumonia 2 months prior to his last admission. The pneumonia was followed by a right hydrothorax, jaundice, and ascites. Laparotomy was performed to establish the cause of the jaundice and ascites. Lymph node and liver biopsies were performed to evaluate the extent of Hodgkin's disease and to prove liver involvement. The patient developed an ascitic fluid drainage from the abdominal wound and signed his release from the hospital. He expired at home 2 weeks later without further treatment.

R. W. (Case 20) was admitted for diagnosis with the symptoms of fever, weakness, weight loss, and ankle edema of several months' duration. Shortly after admission she developed an obstructive type jaundice which was intermittent during 3 weeks of observation. An exploratory laparotomy was performed and a hepatomegaly and splenomegaly found, with multiple small yellow nodules in the liver and spleen. The patient's jaundice deepened postoperatively, followed by a rapidly deepening coma. She expired on the third postoperative day and autopsy confirmed the diagnosis of generalized abdominal Hodgkin's disease.

SURGERY FOR CONDITIONS UNRELATED TO HODGKIN'S DISEASE

Surgical problems unrelated to Hodgkin's disease probably arise in these patients as frequently as in the general populace. The surgeon need not hesitate to undertake in-

dicated surgery because the patient already has a disease which will ultimately prove fatal. Seven of our patients sustained surgery for conditions unrelated to the Hodgkin's disease. All but 1 had uneventful postoperative recoveries. One patient expired following her third major operation. Case examples of these surgical problems are presented in the following.

F. P. (Case 24) developed bilateral salpingitis 2 years after the onset of Hodgkin's disease and a bilateral salpingectomy was performed without complication. Twelve years later this patient developed right lower abdominal pain and partial intestinal obstruction approximately 1 week after the institution of nitrogen mustard therapy for an exacerbation of Hodgkin's disease. During the 5 days after the onset of pain, congestive heart failure, left lower lobe bronchopneumonia, and atelectasis developed with a temperature elevation of 105 degrees F. X-ray studies, including a barium enema, showed a perforation of the cecum and a retrocecal abscess. Drainage of this abscess was performed and was followed by clinical recovery. Eighteen months later intestinal obstruction recurred concomitantly with an exacerbation of generalized Hodgkin's disease. No remission of Hodgkin's disease could then be induced by either x-ray or nitrogen mustard therapy. Since intestinal intubation was ineffective in relieving the obstruction, an exploratory laparotomy with lysis of adhesions was performed. The patient expired the following day.

J. L. (Case 25) who had been treated with x-ray and nitrogen mustard therapy for a relatively chronic type of Hodgkin's disease for approximately 3½ years developed signs and symptoms suggestive of acute appendicitis. Point tenderness in the right lower abdomen was in the center of a wide area of x-ray induced induration. Incision was made medial to this area of induration and an acutely inflamed appendix removed without difficulty. This patient had a normal postappendectomy course and has continued to experience remissions induced by therapy as successfully as before surgery.

SURGERY FOR DEFINITIVE TREATMENT OF HODGKIN'S DISEASE

The exact place which surgery holds in the treatment of Hodgkin's disease is difficult to determine. One of our patients had a radical neck dissection for Hodgkin's disease localized to the cervical nodes. This patient is still alive and well after 8½ years, without evidence of recurrent Hodgkin's disease. Similar long survivals have been reported in the literature (3). From this single case we can draw no conclusion.

RESULTS

Thirty-five operations were performed on 31 individuals with Hodgkin's disease. These operations were all major procedures and included 1 radical neck dissection, 6 thoracotomies, 23 laparotomies, 1 mastectomy, 1 drainage of a perirenal abscess, and 3 laminectomies.

In 26 cases (74.3 per cent) the patients were able to leave the hospital following a normal postoperative recovery period. After 21 (60 per cent) of these operations there was a definite improvement in a complication of Hodgkin's disease, thus prolonging life. After 11 operations no definite improvement occurred. Following none of the 26 operative procedures was there a change in the course of Hodgkin's disease; neither acceleration nor retardation of the course was noted.

Our surgical mortality (3 cases) was found only in those cases with either a rapidly progressing disease or a terminal, generalized Hodgkin's disease. Following 26 of the 35 surgical procedures, the patients recovered sufficiently to be discharged from the hospital. Readmissions in each case were required for treatment of the Hodgkin's disease only. The postoperative recovery in the majority of these cases was comparable to that which would be expected in patients without any underlying disease.

The average duration after the onset of symptoms of Hodgkin's disease and prior to surgery in this series of 31 patients, was 26.7 months. The average duration of Hodgkin's disease after surgery was 21.3 months for all cases and 27.7 months if those patients expiring within 2 months after surgery (terminal cases prior to surgery) are excluded. These figures lead one to conclude that the course of Hodgkin's disease is not accelerated measureably by major surgery.

SUMMARY

Patients with Hodgkin's disease tolerate major surgical procedures well and a relatively normal postoperative recovery is to be expected except in those cases in which the Hodgkin's disease is generalized or terminal. Major surgery does not accelerate the course of Hodgkin's disease.

The most frequent complication of Hodgkin's disease requiring surgical intervention in our series has been hypersplenism. Patients with Hodgkin's disease and an associated hypersplenism are usually good surgical risks; postoperative recovery should be rapid and uncomplicated; and correction of this complication by splenectomy undoubtedly prolongs life in patients with Hodgkin's disease.

Exploratory laparotomy and thoracotomy are well tolerated in patients with early Hodgkin's disease; and these procedures are indicated to establish the diagnosis which cannot be established by peripheral lymph node biopsy.

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RECURRENCE AFTER INGUINAL HERNIORRHAPHY

THE operation for the repair of an inguinal hernia is one of the commonest operations done in both large and small hospitals in this country. In teaching hospitals and in other hospitals it is one of the first major procedures attempted by the young surgeon, all too frequently without proper supervision. Many variations in the technique have been offered with the common purpose of eliminating the hernial sac and repairing the weakness. Despite the contributions of many of the most illustrious names in surgery the operating technique is far from standardized. For the indirect hernia high ligation and removal of the sac are almost universally accepted principles. In addition many authors have advised palpation through the internal ring before the sac is ligated. This maneuver will demonstrate an unexpected direct hernia and determine the necessity for a repair of the inguinal floor. Unfortunately so much emphasis has been placed on the methods of repair for the floor of the inguinal canal that many patients who need only a stitch or two accurately placed at the internal ring receive an extensive operation to strengthen an already adequate inguinal canal. This unnecessary procedure that disrupts normal anatomy may be the cause of the next recurrence, particularly if the skill with which the operation is performed is no greater than the judgment that initiated it. Plication or removal of the sac of the direct hernia may be followed by any of the many procedures described to rebuild the inguinal floor.

The success of any operation may be judged by the rate of recurrence, and quite justly so. Many of the results published are based on 3 to 5 year follow-ups. Unfortunately there is adequate evidence to show that an additional 25 per cent of recurrences may be expected 5 to 10 years after operation. Therefore a 10 year follow-up should be the minimum acceptable. Whether a repair that has remained solid for 9 years can be justly criticized when it breaks down 10 years after operation can be questioned. However, any hernia appearing at the site of the previous herniorrhaphy is generally accepted to be a recurrent hernia. At the present time a 3 to 6 per cent recurrence rate for indirect hernias and a 5 to 11 per cent recurrence for direct hernias might be a fair average if based on a 10 year follow-up. The exceptions to these figures are those offered in support of a new technique. So many variations in technique have been offered, backed by superior results, that one cannot help but question if the better results are due to the new technique advocated or to the fact that the author has made himself a superior surgeon in the repair of hernias.

Factors which influence the recurrence rate have been carefully studied. The relative importance of age, obesity, habitus of the patient, concomitant disease, postoperative complications, early ambulation, as well as the choice of suture material and anesthesia, have all been evaluated. Regardless of these factors the basic cause of recurrence is generally agreed to be in the surgical technique employed. Errors in the surgical technique have been postulated to explain recurrences. Failure to find or properly remove the indirect sac is the commonest excuse offered for recurrence of an indirect inguinal hernia. Certainly the importance of this step must be granted. However, one can be sure that all surgeons with adequate experience in this field have been faced with the unpleasant fact of a prompt recurrence of an indirect inguinal hernia after they personally had removed the sac. This has even been reported when the sac was invaginated and sutured to the abdominal wall. The answer is that the internal ring was not accurately closed.

Recurrences of direct hernias have been explained by failure to recognize the direct hernia or failure to employ the then advocated technique to strengthen the floor of the inguinal canal. Regardless of extenuating circumstances it is generally agreed that recurrence is evidence of an error in the surgical technique employed. This being so it is strange that so little attention has been directed toward the recurrence itself, which is the evidence per se of the error. This may be explained by the fact that recurrences have generally been described to fit into the established classifications of hernia rather than being described as an anatomical defect that resulted from the previous surgery. The lack of more specific information may also be explained by the poorly written operative note, sometimes dictated by an assistant who did not have an opportunity to see the defect and to describe it accurately. Therefore the majority of reports deal only with "direct" or "indirect" recurrences.

Those reports which are based on a careful study of the recurrences found at reoperation give more specific information than implied by the terms "direct" and "indirect." In one report slightly over half the recurrences were in the region of the internal ring but many of these were small defects requiring only one or two stitches to close. The rest of the repair was solid. In an additional 20 per cent the defect was localized to a small point at the pubic tubercle. The floor of the inguinal canal and internal ring were still solid. Therefore repair of the floor of the inguinal canal was at fault in the remaining 25 per cent. This might indicate that more attention should be given to the ends of the inguinal canal and less emphasis placed on the repair of the floor. That is to say that the evidence points toward an error in closing the internal ring or in starting the repair at the pubic tubercle in approximately 75 per cent of these recurrences. Although the vast majority of the "classic descriptions" of hernia repair are quoted for their method of repairing the floor yet they all emphasize these two points, i.e. an accurate closure of the internal ring and proper attention to the first stitch at the pubic tubercle. A re-emphasis of these principles would seem indicated. It is interesting to note that in those patients reoperated upon soon after the recurrence it is rare to find an incisional hernia along the suture line of the floor. Also it is unusual for a femoral hernia to occur under the inguinal ligament despite the disrepute into which this ligament has fallen.

The objective in considering any problem is to stimulate interest and improve upon the previous results. Further study of the problem of recurrence after inguinal herniorrhaphy

should be directed toward the recurrence itself. Early reoperation for the recurrence will give a true picture of the failure as well as allow the repair of a small defect instead of a complete hernia. Accurate anatomical descriptions of the recurrence rather than an attempt to fit the recurrence into the accepted classification of hernias will point to the errors to be corrected. For the benefit of the surgeon unable to profit by his own errors further reports are needed that accurately describe the recurrence seen after inguinal herniorrhaphy. E. THURSTON THIEME.

THE IMPORTANCE OF SPINAL FLUID ANALYSIS AND CONTRAST MYELOGRAPHY WHEN PROTRUDED LUMBAR DISC IS SUSPECTED

IN the study of the patient who is suspected of having protrusion of a lumbar intervertebral disc it is important to carry out intraspinal investigation as well as to obtain a careful history and to do a general physical examination, a neurologic examination, an orthopedic examination, and a roentgenologic examination of the lower thoracic, lumbar, and lumbosacral regions. Intraspinal investigation involves spinal puncture with manometric studies, examination of spinal fluid, and contrast myelography with either air or iodized oil.

Some surgeons operate on a number of their patients who have low back and sciatic pain without employing intraspinal studies, reserving these studies for the cases which they consider to be atypical. In employing this practice, these surgeons are probably influenced by the fact that 90 to 95 per cent of all protrusions of lumbar discs occur at either the fourth or fifth lumbar interspace. The practice of these surgeons who operate "directly" on patients presenting so-called typical signs and symptoms of protrusion of the fourth or fifth lumbar disc is to explore the fourth and fifth interspaces in these cases.

There are four reasons why it is deemed advisable to do intraspinal investigations on all patients suspected of having a protruded lumbar disc: (1) Tumors of the spinal cord in, and cephalad to, the lumbar region may simulate the signs and symptoms of a protruded intervertebral disc. (2) In some instances, protrusion of the third, second, or first lumbar intervertebral disc may produce signs and symptoms commonly seen in protrusions occurring at lower intervertebral levels. (3) Multiple protrusions occur in about 18 per cent of all cases of protrusion of an intervertebral disc, and since they may involve contiguous or scattered intervertebral spaces, contrast myelography is the only practical method by which they may be accurately localized prior to operation. (4) In rare instances, a protruded lumbar intervertebral disc and a tumor of the spinal cord may coexist, giving rise, usually only to so-called typical signs and symptoms of protruded disc. (5) When contrast myelography is employed, the lesion is accurately localized in a high percentage of cases; thus the necessity of entering normal interspaces is obviated.

In a recent analysis of 1,245 surgical cases of protruded lumbar disc, and 51 cases of spinal cord tumors which arose at or below the level of the tenth thoracic vertebra and which were operated on during the same interval, Craig, Svien, Dodge, and Camp[1] found that in 20 cases of spinal cord tumor the clinical diagnosis prior to intraspinal investigation was protrusion of an intervertebral disc at the fourth or fifth interspace. In 7 additional cases, the clinical diagnosis of "protruded disc

[1]CRAIG, W. MCK., SVIEN, H. J., DODGE, H. W., JR., and CAMP, J. D. Unpublished data.

versus spinal cord tumor" was made. The reasons for adding the alternate diagnosis of spinal cord tumor in these 7 cases were: (1) a history of pain at night, (2) the presence of marked scoliosis or kyphoscoliosis, and (3) a history of malignant disease elsewhere in the body. The signs and symptoms in all 27 cases strongly suggested the presence of a protruded disc at either the fourth or fifth interspace, yet in only 5 were the tumors located in the region of the fourth or fifth lumbar vertebra, the lesions in the remaining 22 cases being located cephalad to the fourth lumbar level. Thus, had "direct" operation without intraspinal investigation been carried out in the entire series of 1,245 cases, it seems reasonable to suppose that the spinal cord tumor would have been missed in at least 22 instances. There were 3 instances in which an operation performed in another community for a protruded disc had not resulted in relief of symptoms and in which subsequent intraspinal investigation revealed the presence of spinal cord tumor. In 2 of these cases, spinal fusion had been carried out at the time of the operation for protruded disc.

The importance of recognizing the presence of a spinal cord tumor in such cases as those under consideration is emphasized by the fact that in 19 of 27 cases the tumors were such that they could be completely removed and the patient cured. The loss of time occasioned by operation for protruded disc in the presence of unsuspected tumor of the cord could well have resulted in irreparable damage to the cord in some of these cases.

In the series of 1,245 cases of protruded lumbar disc, there were 26 cases in which protrusions cephalad to the fourth lumbar interspace existed either alone or in combination with protrusion at the fourth or fifth interspaces but in which the history and the neurologic, orthopedic, and plain roentgenographic examinations gave no hint of protrusion of a lumbar disc at a higher level; only a strong indication of the presence of a protrusion at either the fourth or fifth interspace was evident. In 10 of these cases, the surgeon whose practice it is to explore directly the fourth and fifth interspaces would have had the false security of finding a protruded disc at either the fourth or fifth interspace, but he would not have relieved the patient of his symptoms because a protrusion at a higher level would have been missed.

Cases in which protruded lumbar intervertebral discs coexist with spinal cord tumors in the region concerned, although extremely rare, do exist. Cases of this type have been reported by Camp[2], and additional cases are currently being reviewed by Camp and Betts[3]. Although the number of such cases in which the signs and symptoms point to a diagnosis of protruded intervertebral disc is not large, it is only by intraspinal investigation that the presence of both lesions can be determined.

These findings suggest, then, that even if a careful history is taken and careful general, neurologic, orthopedic, and simple roentgenographic examinations are carried out in cases of suspected protruded lumbar intervertebral disc, lesions responsible for the patient's signs and symptoms will be missed at operation in a significant number of cases unless examination of the spinal fluid and contrast myelography or differential spinal puncture are carried out routinely in every case. For this reason and to obviate exploring normal interspaces intraspinal investigation seems to be justified in all such cases.

HENDRIK J. SVIEN
HENRY W. DODGE, JR.
JOHN D. CAMP

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THE CURSE OF CAIN

A. L. HERRING, JR., M.D., and ROBERT W. PRICHARD, M.D., Boston, Massachusetts

"I am here in despair, and why? For doing what Brutus was honored for—what made Tell a hero. And yet I, for striking down a greater tyrant than they ever knew, am looked upon as a common cutthroat. . . . I do not repent the blow I struck; I may before God, but not to man. I think I have done well. Though I am abandoned with the curse of Cain upon me, when, if the world knew my heart, that one blow would have made me great, though I did desire no greatness."

THESE words were scribbled in his "diary" by the most famous assassin in American history, John Wilkes Booth. He wrote them as he lay in hiding near the Potomac River in Charles County, Maryland. The date was April 21, 1865, and seven harrowing days had passed since he had fired through Abraham Lincoln's head the shot that started one of the most chaotic fortnights the country has known.

In the rush of events which stemmed from the tragedy in Washington the medical profession figured in many ways. Before these interesting facets of the tragedy are traced, it may be well to review the assassination briefly.

On several occasions during the Civil War, attempts were made to abduct President Lincoln, all of them the actions of individuals without official sanction from the Confederacy. Among these plans was one which nearly succeeded, conceived by the popular young tragedian, John Wilkes Booth. The actor employed an ample staff for the purpose but their attempts were narrowly thwarted by chance on several occasions. After Lee's surrender to Grant on April 9, 1865, the assassins grew desperate. Booth quickly laid a plan to assassinate Lincoln, based on the knowledge that he would appear at the performance of a popular comedy then playing at Ford's Theater in Washington.

From the Laboratory of Pathology, New England Deaconess Hospital.

Booth's professional tours had familiarized him with Ford's Theater and the situation was one which appealed to his dramatic spirit. Having obtained a cue from the action of the play during its morning rehearsal, Booth entered Lincoln's box at the moment when he knew that the stage was relatively clear. With a single shot Derringer he sent a ball into the President's head. Turning, he slashed the arm of Major Rathbone who was in the President's box, and leaped over the edge of the box onto the stage 12 feet below. Ironically, his spur caught in an American flag draped over the front of the box and the actor struck the stage off balance, fracturing his left ankle. As he hurried through the wings two men ineffectually clutched at him, but he eluded them and mounted his horse in the alley behind the theater.

As Booth left the city he was joined by a former drug clerk, "Davie" Herold, and together they rode toward the lower Potomac through country thoroughly familiar to Herold. As the night wore on Booth became increasingly disabled by his injury and realized that he must seek medical aid. From an excursion through the same country the preceding autumn he knew of Dr. Samuel Mudd, who was a country doctor with Southern sympathies, and he determined to reach his house.

At 4:00 a.m., nearly 6 hours after the shooting, Booth and Herold awakened Doctor Mudd. Booth wore a false beard and disguised his voice, introducing himself as Tyler, and his companion as Tyson. After a cursory examination the doctor advised his patient that he would have to halt in his journey. Booth was carried to an upstairs room by the doctor and Herold. There Dr. Mudd cut away the assassin's cavalry boot and examined the injury. He later gave his diagnosis as, "The outward bone was broken at nearly right angles across the limb, about two inches above the instep."

On the morning following the assassination Booth and Herold left the doctor's house while Mudd was making his daily rounds. Then followed six days and nights of hiding on the Maryland side of the Potomac awaiting a chance to cross to the Virginia side. A manhunt of unprecedented size was in progress. Finally, Booth and his companion were cornered in the Garrett barn near Bowling Green, Virginia. Herold surrendered and Booth was shot, either by his own hand or that of a soldier in the posse which discovered them. Booth did not die immediately but lingered for a short time and expired at dawn on April 26, 1865, twelve days after the shooting of Lincoln.

The story now returns to Washington on the night of the assassination. Attempts were made on other lives than Lincoln's that evening. Seward, the Secretary of State, was assaulted and saved by a stroke of fortune only. His would-be assassin's blade had been turned by a metal collar which had been applied by Seward's physicians as part of a cumbersome fixation apparatus for the secretary's recently fractured jaw. An assassin went to Vice-President Johnson's hotel, but he lost his courage and got only as far as the bar. Stanton, the Secretary of War, claimed to have been mysteriously threatened. Another assassin was reportedly aboard General Ulysses Grant's train. The telegraph lines leading into and out of Washington were interrupted for hours by unknown agencies. The Capitol experienced a tremendous panic, with reports rife that it was about to be invaded, sacked, and burned.

LINCOLN'S LAST HOURS

The stunned silence that followed immediately after Booth's shot was punctuated only by the screams of Mrs. Lincoln. A young army surgeon, Charles A. Leale, was the first physician to reach Lincoln's side and he was soon assisted by others. The President's head had slumped onto his chest and a small amount of blood had formed a clot on his left shoulder. Neither pulse nor respirations could be detected. A rapid examination by the doctor showed no external wound, but upon lifting Lincoln's eyelids Dr. Leale saw unmistakable signs of intracranial injury and soon found the bullet hole behind the left ear. At once, Leale and his assistants began artificial respiration, obtaining a quick but feeble response.

The attending physicians felt that the President should be moved as short a distance as possible, and he was carried across the street to a boarding house, seated upright in the rocking chair that he had occupied when he was shot. Carrying the chair were Capt. Oliver Gatch and his brother, Dr. Charles Gatch, respectively the father and uncle of Dr. Willis Gatch, the inventor of the Gatch bed and present professor of surgery at the University of Indiana.[1] Mustard plasters, hot water bottles, and whiskey were used, the latter until the President became unable to swallow. The left side of the head was kept in the dependent position and clots which formed at the wound of entry were removed from time to time. Dr. Leale turned his illustrious patient over to Lincoln's family physician, Dr. Stone, and to Surgeon-General Joseph K. Barnes of the Army. The bullet was searched for with fingers and probes, to no avail, although the piece of bone driven into the brain by the bullet was removed. Seven hours after the shooting the irregularity of the President's pulse and respirations presaged imminent death. At 7:21 and 55 seconds Dr. Charles Taft recorded the cessation of Lincoln's respirations, and at 7:22 and 10 seconds the pulse became undetectable. Five hours after Lincoln's death, an autopsy was performed in the White House. Only the cranial cavity was opened. The findings are presented in the report which is reproduced below:

"Surgeon General's Office
Washington City, D. C.
April 18th, 1865.

"Brig. Gen. J. K. Barnes
Surgeon General, U. S. A.
General:

"I have the honor to report that in obedience to your orders and aided by Asst. Surg. E. Curtis, U. S. A. I made in your presence at 12 o'clock this morning an autopsy on the body of *President Abraham Lincoln*, with the following results.

" "The eyelids and surrounding parts of the face were greatly ecchymosed and the eyes somewhat protuberant from effusion of blood into the orbits.

"There was a gunshot wound of the head around which the scalp was greatly thickened by hemorrhage into its tissues. The ball entered through the occipital bone about an inch to the left of the median line and just above the left lateral sinus, which it opened. It then penetrated the dura mater passed through the left lateral ventricle and lodged in the white matter of the cerebrum just above the anterior portion of the left corpus striatum where it was found. The wound in the occipital bone was quite smooth, circular in shape with beveled edges, the opening through the internal table being larger than that through the external table. The track of the ball was full of clotted blood and contained several little fragments of bone with a small piece of the ball near its external orifice. The brain around the track was pultaceous and livid from capillary hemorrhage into its substance. The ventricles of the brain were full of clotted blood. A thick clot beneath the dura mater coated the right cerebral lobe. There was a smaller clot under the dura

[1]Dr. Gatch, in a personal communication, says that Lincoln was removed from the theater because public sentiment against theater-going was so great that it was feared that Lincoln's death in a theater would be considered a national disgrace. Dr. Gatch did not mention the fact that it was also Good Friday.

mater of the left side. But little blood was found at the base of the brain. Both the orbital plates of the frontal bone were fractured and the fragments pushed up towards the brain. The dura mater over these fractures was uninjured. The orbits gorged with blood.'

"I have the honor to be
Very Respectfully
Your obt Servt
(signed) (Signed) J. J. Woodward
Asst Surgn. U.S.A.

A true copy
(signed) George Otis
Assistant Surgeon U. S. A."

Dr. Curtis remarked to his mother, in a letter written a week after the autopsy, that he was surprised to find that the great man's brain weighed no more than that of an ordinary mortal. He also stated, "I was simply astonished at the showing of the nude remains, where well-rounded muscles built upon strong bones told the powerful athlete. Now did I understand the deeds of prowess, recorded of the President's early days."

Despite the exact statements of Dr. Woodward's autopsy report, another observer who was present at the autopsy wrote that the bullet had lodged on the opposite side from that reported above. This discrepancy has never been completely explained, but it has little bearing on the case. Another disputed point is the exact origin of the force which shattered both of the President's orbital plates.

The disgraceful "Conspirator's Trial" which followed Lincoln's assassination is a blot on American judicial history. Deprived of revenge upon the chief conspirator, the outraged Government descended on the eight suspects in its grasp. These prisoners were tried by an irregularly constituted military tribunal, with no regard for due process of law. Four of them went to the gallows in what was termed at that time "judicial murder." Among the four sentenced to life imprisonment at hard labor was Doctor Mudd.

SAMUEL MUDD, M.D.

Samuel Mudd, a native of Maryland, attended the University of Maryland Medical School. His practice in the countryside around Bryantown, a sleepy agricultural community then as now, was a leisurely one, and he devoted much of his attention to farming. Prior to the Civil War Dr. Mudd was a slave owner and like many another whose substantial investment was lost with the Emancipation Proclamation, Mudd sympathized with the South, but not actively. In the Fall preceding Lincoln's assassination Booth passed through Bryantown and spent a night at Mudd's house. The trip was disguised as a horse-buying expedition, but its real purpose was the scouting of a route over which Lincoln could be transported to Richmond after his projected kidnapping. Mudd is said to have been unaware of this ulterior motive. In the half year which elapsed until Booth's return to Mudd's house the doctor is said to have called on Booth in his Washington hotel, for purposes which were unknown, some time in January, 1865.

As already outlined, Booth arrived at Mudd's home in disguise, and although the hour was late and the injury unusual, the doctor can be excused for not suspecting the true state of affairs. However, he made no attempt to report his mysterious patient to the authorities for three days, despite the fact that the countryside was alive with news of the assassination and troopers were combing the roads. The disappearance of the injured man should have further aroused his suspicions, no matter how obtuse he was. When Mudd stood before the bar of justice it was difficult to convince the commission that his relationship with Booth had been merely that of doctor and patient. His lawyers stated that he was a suspicious and taciturn man who would not confide even in his counsel. It is conceivable that the retiring and resentful country practitioner reasoned just as a reporter of the day did when he put the following words in Mudd's mouth. With the contemporary journalistic disregard for truth he had Mudd say, "At last they are gone . . . and I am confoundedly glad of it. I don't much like to have a finger in this devil's pie. I am rejoiced that I was not in Washington when the deed was done. However, I have made $25.00 by the operation and I don't think they will dare to hang a man for doing a job in the way of his profession."

Together with three other hapless people linked to Booth, Mudd was sentenced to imprisonment at Fort Jefferson in the Dry Tortugas, an island in the Florida Keys. An account of Mudd's life was written by his daughter, Nettie, and includes his letters home during his imprisonment. In reading his letters one captures his feeling of utter frustration, despairing loneliness, and righteous indignation. He was treated with no consideration for his former status as a professional man and he was forced to work as a common laborer. After one nearly successful attempt to escape, he settled down and became a model prisoner. His letters contain a few interesting medical items. On July 8, 1867 he mentioned "millions of little mosquitoes that are very annoying." On August 25 there was a death from yellow fever, and a few days later having volunteered his services, he was laboring selflessly to fight a growing epidemic. He notes

that he had some success in checking the spread of the disease by placing all patients on the downwind side of the island. His reason for doing this was that he thought the constant trade winds would carry the infectious vapors and miasmata off to sea. Had he appreciated the identical effect of the wind on the mosquitoes, he might have anticipated the work of Walter Reed, then a boy of sixteen. Mudd's letters of this period contain many observations on the clinical course and pathology of yellow fever, a disease with which the Maryland physician had had a scanty previous experience. Mudd stressed the importance of vigorous treatment from the very outset, and frequently referred to the importance of good nursing care. Proving that psychosomatic medicine was understood many years ago, he stated, "It was not long before I discovered I could do more with nine cases out of ten by a few consoling words than with all the medicine known to me in the materia medica."

After four years' imprisonment Dr. Mudd was released by President Johnson's order and returned home. His health was broken and financially he was almost ruined.[1] He died fourteen years after his release at the age of fifty, on January 10, 1883. The ever unfortunate doctor had contracted pneumonia on a wet night when he had ventured out to attend a patient.

The weight of evidence suggests that Mudd was not an active participant in the plots against Lincoln, either in the kidnapping or the murder. His chief crime lay in his failure to report the presence of suspicious characters in his house, admittedly a large indictment. However, he made no attempt to conceal the visit by Booth or the treatment of his injuries. When investigating detectives came to Mudd's house the doctor himself showed them the boot he had cut from Booth's injured leg. This was later said to match the one on the assassin's other leg at the time he was killed.

BOOTH'S FINALE

Philip Stern has pointed out that the central figure in the Lincoln assassination was not the Great Emancipator but the 26 year old actor, Booth. This pathetic man's background does not contain any of the usual influences said to mold a killer. Booth was a gifted member of a famous theatrical family and had enjoyed great independent success on the stage. Although overshadowed by his famous older brother, Edwin Booth, he was gaining yearly in eminence. John Wilkes had never known poverty. At the time of the assassination he was making roughly $20,000 a year from his acting alone and he invested his money wisely. Critics said he was at his best in Shakespearean tragedies. His personal beauty and charm were universally dwelt upon by all who knew him, men and women alike. His dashing airs, classic features, and dramatic ability lead to his description as a combination of Lord Byron and Edgar Allan Poe. George Alfred Townsend said of him in 1865, "He possessed a fine Doric face, spare at the jaws and not anywhere overripe, but seamed with a nose of Roman model, the only relic of his half-Jewish parentage, which gave decision to the thoughtfully stern sweep of two direct, dark eyes, meaning to women a snare, and to men a search warrant." His love affairs were numerous, and his pockets contained pictures of four women at the time of his capture. He was engaged to a fifth woman and was rumored married to yet another. His brother Edwin said he was possessed by quixotic notions. Perhaps his senses, sharpened to the dramatic and misguided by heated notions of Lincoln's character, truly rendered the man insane. However, examination of his actions gives the picture of a man in full possession of his faculties, acting with the misdirected zeal of any fanatic. Booth is worthy of more study than the common opinion of him as a drink-crazed madman inspires.

When Booth was carried from the barn on the Garrett plantation, as previously outlined, he was fatally wounded; history supports that much. Whether the mortal wound was self-inflicted or was the work of a fanatically religious soldier, Boston Corbett, is a moot question. After the shooting in the barn Booth was laid on the floor of the farm house porch and was revived by a dash of cold water. A bullet wound was present high on the right side of the neck and a wound of exit was low on the left side. The victim could breathe and talk feebly but was quadriplegic. The tragedian suffered intensely in his last act, and begged his captors to kill him. Several times he asked that his arm be lifted, apparently to see if it was still attached. As secretions accumulated in his throat, Booth seemed unable to raise them. He was concerned over this and thought that he was bleeding into his throat. Colonel Conger, in charge of the capturing group, told him to open his mouth and reported that he could see no blood. At sunrise, on April 26, Booth asked the Colonel to tell his mother ". . . I died for my country," and then expired. The body was sewn in a blan-

[1]The plight of the doctor has led some people to state that the familiar expression "His name is mud!" stemmed from the events cited. Although the supposition is justified, there is no support in fact. Partridge's "Dictionary of Slang and Conventional English" reveals that the expression was in use before Samuel Mudd was born.

ket, thrown on a farm wagon, and placed aboard the monitor "Montauk" for transportation back to Washington.

The identification of Booth's body when it was brought to the Navy Yard in Washington was badly handled, and this has resulted in the perpetuation of numerous myths stating that Booth was not slain on the Garrett farm. Over the years several individuals have claimed to be Booth, and a mummified corpse made the rounds of carnival sideshows for several decades, displayed as that of Lincoln's assassin. The men who brought Booth to bay were accused of fabricating the entire episode in order to collect the large rewards offered. Others have claimed that Booth was but one of a group which plotted to kill Lincoln and that the others were anxious for a scapegoat, preferably a dead one. This shadowy group is said to have included high Government officials, perhaps Secretary Stanton and Vice-President Johnson themselves.

Booth's body, as it lay aboard the Montauk, was carefully guarded. The Secretary of War, Stanton, had decided that none of Booth's family or close personal friends were to be allowed to view the remains. He felt that the South might attempt to make Booth a martyr. Accordingly, six to nine people, none well acquainted with the assassin, filed by the dead man. Official photographers were summoned but were not allowed to photograph the corpse. All witnesses agreed that the dead man was Booth.

The one witness to whom history has attached most credence was Dr. John Frederick May, a prominent and well-trained surgeon of Washington, a professor at the Medical Department of Columbian University, now George Washington University, and at the University of Maryland. Dr. May wrote an article in 1889 describing his part in the identification of Booth's body. It was titled "The Mark of the Scalpel," and was written in the romantic style of the day. The doctor stated that some years before the assassination he had excised "quite a large fibroid tumor" from the back portion of Booth's neck, on the left. Booth asked Dr. May to tell any inquirer that the operation was for removal of a bullet! Despite Dr. May's injunction that the actor absent himself from the stage until the wound healed, Booth played on, and during an emotional scene an actress disrupted the incision. The wound healed slowly by granulation and left a scar which May claimed was easily remembered. Before viewing Booth's body the doctor detailed his recollection of the scar to the military attendants and states that his description tallied exactly with a scar the

Fig. 1. His name was Mudd.

dead man bore. Dr. May goes on to say that save for the scar, he would not have recognized the assassin, so wasted were his features. Historians have pointed out that Dr. May contradicted himself in his testimony at the time of the identification and in his article, but the contradictions were those of right and left. In consideration of the time elapsed, the statements are understandable errors. The doctor follows his factual account of Booth's identification with an interesting discourse on the identification of bodies and the strength of madmen.

In addition to Dr. May's facts, it was noted that the corpse had a Pott's fracture of the leg, as described by Mudd, wore John Wilkes Booth's vest containing personal items, and had the initials J.W.B. tattooed on its hand. The tattoo was the work of Booth himself and had been done with India ink when the actor was a boy. Doubters of Booth's identity claim that persons wishing to substitute a body for Booth's could have fractured its leg, but witnesses described the broken leg as swollen and blackened; this would not have occurred postmortem. The imposters who have claimed to be Booth over the years have not had the tattoo on the hand as mentioned. The mummified corpse which was displayed as Booth's was

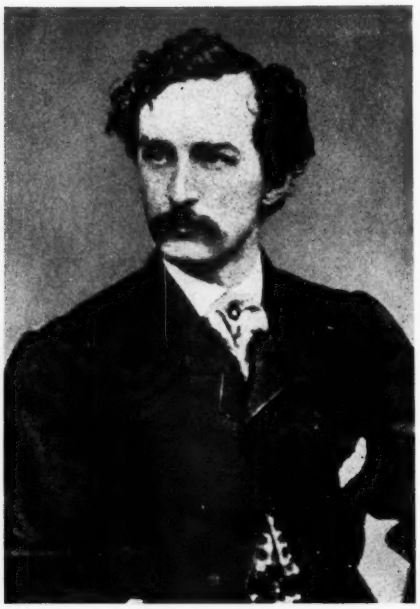

Fig. 2. Tragedian.

x-rayed and the pictures submitted to experts, but they would not commit themselves as to the nature of the injuries shown. This same mummy was examined in the Crime Detection Laboratory of Northwestern University. No scar was found on the mummy's neck, even when it was examined under ultraviolet light.

Those who viewed the body aboard the warship stated that there were freckles across the nose. Booth was noted for his "marble-white" complexion, and these blemishes were cited as evidence against the corpse being Booth. Medical authorities have stated that prolonged sepsis and starvation would not produce such marks. But it is conceivable that during life they were concealed by the actor's make-up, or were unnoticed by his friends. His position during transportation and the role of livor mortis are not clear.

Asia Booth Clarke, Booth's sister, stated that her brother once had a carbuncle on his neck which had been opened during a visit to her home in Philadelphia. This infection also left a scar and has been cited by detractors of Dr. May's testimony as another shadow upon the accuracy of his identification of the body. Yet another possible origin for a scar is found in an account by Townsend of a bandaged Booth telling friends in Richmond before the war that he had been recently stabbed in the neck by a jilted mistress in Montgomery, Alabama.

Booth's body returned for its final identification by his family to a mortuary across the alley from Ford's Theater in which he had made his last appearance as a tragedian. Later, parts of his body were deposited in the theater building itself when that structure became the home of the Army Medical Museum in 1867. A contemporary newspaper stated that Booth's brain and heart, preserved in alcohol, were given to the Army Medical Museum. In fact, Surgeon-General Barnes deposited there only a portion of the spinal cord and the third, fourth, and fifth cervical vertebrae, removed at autopsy. The spinal cord was perforated from right to left, and the laminae of the fourth and fifth vertebrae were fractured by a carbine bullet fired from a few yards distant. Dr. Ruell Sloan, present curator of the Army Medical Museum, who supplied these facts, states that there is no record of Booth's brain and heart ever having been in the possession of the museum.

When Booth's body was exhumed from the courtyard of the Washington Navy Yard in 1869 and turned over to his family as aforementioned, an excellent opportunity again existed for satisfactory identification of the remains. Eisenschiml points out that Booth's uninjured leg still bore the cavalry boot the mate of which had been turned over to the detectives by Dr. Mudd. This piece of footwear could have been matched exactly with the one in possession of the authorities and the question of Booth's identity settled with finality. Unfortunately no positive steps were taken. However, the possibility of the comparison of boots still exists since Booth's body lies in the family plot in a Baltimore cemetery and the boot removed in 1865 is still in the War Department archives. Authorities on leather state that good leather is very resistant to decay. A dentist supposedly identified his handiwork in the disinterred actor's mouth, but he never made a written statement and his name was never disclosed by the family. One of the witnesses present at this event stated that the corpse's hair had grown a foot! One hopes that the rest of the observations were more accurate.

THE MAD HATTER

Boston Corbett, the soldier who claimed to have shot Booth, is a man of medical interest. He was born Thomas H. Corbett in London in 1834, and came to the United States at an early age. His wife and their infant son died in New York City. According to a Sunday School teacher who

wrote a sketch of him, he then "became despondent, and a victim of the cup which destroys body and soul . . .," and moved to Boston where he plied his trade of hat-finishing. Subsequent events convey the impression that he was truly "mad as a hatter." After a religious meeting, Corbett was rebaptized "Boston Corbett" by the Methodist Episcopal Church and began to preach in the streets. After one of his North Square meetings he was accosted by two young women who sought unsuccessfully to inveigle him from the path of righteousness. He then went home, and "to be holy," as he phrased it, performed an operation which is well described in his admission history to the Massachusetts General Hospital on July 16, 1858. It is given verbatim:

"Is a Methodist, and having perused the 18th and 19th chapters of Matthew, he took a pair of scissors, and made an opening one inch long in the lower part of the scrotum. He then drew down the testes and cut them off. He then went to prayer meeting, walked about some, and ate a hearty dinner. There was not much external hemorrhage, but a clot had filled the opening so that the blood was confined in the scrotum, which was swelled enormously and was black. He called on Dr. Hodges, (R. M.), who laid it open and removed the blood; he tied the cord and sent him here."

Then followed concise progress notes indicating an uneventful course. It is an interesting commentary on the induction physical examinations of the day that Corbett was later admitted to the Army. During his service career he was frequently in difficulty because of his religious fanaticism. At the time he shot Booth, in direct disobedience of the standing order for the capture of the assassin alive, he claimed he did so on divine instruction. Having collected about $2,500 of reward money for his part in Booth's capture, Corbett appeared once more in history, this time as a sergeant-at-arms in the Kansas legislature. One day he took issue with the discussion on the floor and adjourned the legislature at gun point. For a short period thereafter he was in an insane asylum but escaped and disappeared into Mexico. One legend has it that for years thereafter he traveled through the South as a very successful "detail man" for a patent medicine firm.

THE MAN OF MYSTERY

Among those who were said to have conspired with Booth in Lincoln's assassination was a mysterious young man named John Surratt, son of the unfortunate Mrs. Surratt who was hung for supposed complicity in the Presidential murder. It is said that he had been detailed to kill Grant as the General's train rode through the night away from Washington. Whatever his part, he

Fig. 3. The Mad Hatter.

fled to Canada, thence through England to Italy where he became a member of the Papal Zouaves. With him in this colorful organization was an acquaintance from Maryland who betrayed him, seeking the reward offered for this last of the conspirators. After much diplomatic maneuvering Surratt was arrested. While being escorted to a privy under guard he leapt over a railing surrounding a castle courtyard, into an adjoining ravine more than 100 feet deep, emerging uninjured. The mystery of the astounding drop without injury was solved on inspection of the terrain. The castle privies jutted out over the abyss and the waste dropped onto a narrow ledge halfway down. It was in this unsavory material that the escapee found a cushion for his fall. He made his way across Italy, borrowed money from some British people in Naples, and boarded a British freighter for Egypt. An unsuccessful attempt was made to remove him at Malta, and finally, in Alexandria, he was captured through an instrument of the medical profession. In Naples, from which he had come, a cholera epidemic was raging and incoming third-class passengers from that port were quarantined for a week. Standing among the quarantined immigrants in his multicolored uniform, Surratt was easily found by the pursuing authorities and was brought back to the United

States. A famous trial was held, in which Surratt, despite infamous legal tactics by the Government,[1] was not convicted, and he finished his days as a respected shipping clerk in Baltimore. He would never discuss the Lincoln assassination.

MISCELLANY

Interesting miscellany involving the medical profession may be mentioned. Booth's contacts with physicians in his flight did not end with Dr. Mudd. Having crossed the Potomac into Virginia the suffering assassin was tormented by his swollen, blackened leg, and again sought medical aid. A farmer was enlisted to take Booth to a nearby practitioner, a Dr. Stewart. The assassin represented himself as a wounded Confederate soldier. Booth was received with unveiled coolness, since the inhabitants of the countryside were becoming weary of begging ex-soldiers by that time. He asked the doctor to examine his leg. It appears that this country doctor, even at that early date, had become specialized, for he answered, "I'm a physician—not a surgeon. I doubt whether I can be of much assistance to you." The outraged Booth was fed in the kitchen, after which the doctor informed him that he could not accommodate him for the night since the house was already overfull. His specialization and plethora of guests were good fortune for the doctor. He might easily have become professional company for Dr. Mudd in the Dry Tortugas, with the judiciary as volatile as it was at the time. Booth's angry note to the doctor after these rebuffs is a commentary on those who have pictured Booth as a raving maniac. Following the exchange above, Doctor Stewart recommended the strangers to the cabin of William Lucas, a Negro freedman. The note was returned to the doctor by Booth's guide:

"Forgive me, but I have some little pride. I hate to blame you for want of hospitality, you know your own affairs. I was sick, tired, with a broken leg, in need of medical advice. I would not have turned a dog from my door in such a condition. However, you were kind enough to give us something to eat, for which I not only thank you, but on account of the reluctant manner in which it was bestowed, I feel bound to pay for it. It is not the substance, but the manner in which kindness is extended that makes one happy in the acceptance thereof. 'The sauce to meat is ceremony; meeting were bare without it.' Be kind enough to accept the enclosed $2.50 (though hard to spare) for what we have received."

This masterful sarcasm was the work of a man hunted by a nation, eight days with very little food, no shelter and suffering from a broken leg.

[1]Eisenschiml states, "To physicians it was usually intimated by sarcastic cross-examiners that they were abortionists.".

Surgeon-General J. K. Barnes, who was present when Lincoln died, was an active participant in a series of governmental tragedies. After Lincoln's death he assisted in the identification of Booth's body. He was present when Secretary Stanton died. He was one of the staff who attended the assassinated President Garfield, 16 years later, and assisted in the autopsy on him.

The medical profession was represented in the infamous Conspirators' trial in another unusual way. One of the judges, General T. M. Harris, was a West Virginia physician who had fought as a line officer during the Civil War. It was not stated what his qualifications for the judiciary were. At the outset of the trial he raised a question concerning one of the defense lawyers which involved minor political and legal matters, and referred to an incident which had occurred while he was fully occupied in the field during the war. This has led one commentator to deduce that the words were put in the doctor's mouth, implying that the doctor had no business in a judge's seat. Certainly his presence there was but one of many incongruities.

The curse of Cain had not ended for Ford's Theater. After the Army Medical Museum was moved to a special building on the grounds of the Smithsonian Institution, the old theater was once again the scene of sudden death. On June 6, 1893, while being used as a storehouse for the Adjutant-General's records, the building collapsed killing 22 Civil War veterans.

The other occupants of Lincoln's box also felt the curse. Major Henry Rathbone, the Army officer who was stabbed by Booth in Lincoln's box, was one of the last to die. Rathbone married the lady he escorted on that fateful evening and two children were born of the union. During a trip to Germany with his family the Major suddenly went berserk, killed his wife, and attempted suicide. He died in a German insane asylum in 1911.

This brief review has attempted to show the part played by the medical profession, willingly or unwillingly, in this great American tragedy. The writings of lay historians pose questions of medical detail to physicians; it is hoped this review sheds light on these blanks in the matter of Lincoln's assassination.

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REVIEWS OF NEW BOOKS

IN Volume 1 of *Post-Graduate Lectures on Orthopedic Diagnosis and Indications*,[1] Dr. Steindler has not attempted to cover technical procedures, but has in his usual exhaustive manner presented his material with the clinical situation as the basis for multiple case presentation.

This volume is subdivided into two sections. The first section deals with fundamentals which orient the reader as to the sphere of normal and pathological phenomena of the locomotor system and shows the gradual transition between the two.

A great bulk of the material in this section is a condensation of his previous well known work on locomotion. It deals mainly with body symmetry joint motion, and gait. Included is an excellent chapter on contractures with differentiation between myogenic, desmogenic, and dermogenic factors involved. Pain in orthopedic conditions is likewise well covered from the points of view of quality, means of production, duration and intensity, and distribution. Steindler brings out the important difference between referred and reflex pain. Special clinical pain patterns are briefly outlined.

The second section of Volume 1 covers congenital deformities and disabilities with a review of the various theories of their pathogenesis and the conclusion that certain deformities fit with each theory, and that at least we are able to localize most deformities to a specific phase in the life of the embryo.

For the most part all subjects in addition to being well outlined as to etiology, pathology (microscopic, x-ray, and clinical), and treatment are well augmented by photographs of patients and x-ray and microscopic plates.

In his treatment of the chondrodystrophies or disturbance of enchondral bone growth, Steindler classifies the cases in either typical or atypical achondroplasia. The latter group includes the more obscure Ollier's and Marquio's diseases. Madelung's deformity, stippled epiphyses, and multiple congenital exostosis.

The dysostosis group is based on a deficiency of membranous bone formation and the osteopsathyrosis cases show inhibition of periosteal and endosteal growth.

The subjects of giantism and arthrogryposis are also discussed in this section. The remainder of the text is occupied with the presentation of more common deformities specifically located in the spine and thorax, viz., congenital clubfoot, dislocation of the hip, the upper, and the lower extremities.

The material is well presented and attractively edited. Voluminous references occur at the end of each lecture. Some references refer to works well over two centuries old, but one notes a paucity of references to writings of the past decade.

This book is excellent reference material for every student in orthopedics. E. J. CUMMINS.

[1] POST-GRADUATE LECTURES ON ORTHOPEDIC DIAGNOSIS AND INDICATIONS. By Arthur Steindler, M.D., F.A.C.S. Vol. 1. Springfield, Ill.: Charles C Thomas, 1950.

THE *Transactions of the XIIth British Congress of Obstetrics and Gynaecology*, edited by Alec W. Bourne, F.R.C.O.G., and Professor W. C. W. Nixon[2], is an excellent review of the best in British thought on a number of pertinent gynecological and obstetrical problems.

The first session of the meeting, devoted to a consideration of cesarean section, comprised approximately one-third of the volume. Particularly interesting is C. MacIntosh Marshall's statistical comparison of 7,762 cesarean sections performed between 1943 and 1947 with a similar study by Munro Kerr and Eardley Holland for the years 1911 to 1920. The paper by Frank Stabler on cesarean section in infected cases is challenging in its implications.

To this reviewer the general discussion urging a reduction in the number of cesarean sections was not as vital as R. Francis Matters' remarks recommending an increasing appreciation of fetal indications and a consideration of the seriousness of subsequent maternal traumatic states in deciding upon abdominal delivery.

Subjects discussed in other sessions included essential hypertension in pregnancy, chronic hypertension in pregnancy, the management of pregnancy in diabetes, carcinoma of the cervix, with a consideration of cytologic diagnosis of uterine cancer, precancerous changes in the cervix, and the surgical treatment of cervical cancer.

The guest paper, "Endometriosis," by J. V. Meigs of Boston, was a succinct résumé of the subject. Dr. Meigs again presented the interesting hypothesis that endometriosis is the result of delayed marriage and childbearing.

JOHN W. HUFFMAN.

THE third edition of the popular monograph *The Normal Encephalogram*[3] has been revised by Dr. Davidoff to include current knowledge in this field. The material is inclusive, well organized, and generously illustrated. As a result one finds the book easy to read.

The student entering the field of roentgenology, neurology, or neurosurgery will find this monograph an invaluable introduction to the important field of

[2] TRANSACTIONS OF THE XIITH BRITISH CONGRESS OF OBSTETRICS AND GYNAECOLOGY. Edited by Alec W. Bourne, F.R.C.O.G., and Professor W. C. W. Nixon. London: Austral Press, 1950.

[3] THE NORMAL ENCEPHALOGRAM. By Leo M. Davidoff, M.D., and Cornelius G. Dyke, M.D. 3d rev. ed. Philadelphia: Lea & Febiger, 1951.

encephalography and a sound basic framework upon which to build his future experience in this field.

Contained in the first chapter entitled "General Considerations" he will find interesting and valuable information which he should know, but which he would have difficulty in finding elsewhere. Such aspects are the historical, the technique of encephalography, the physical factors, the position of the patient, absorption of the gas, failure of the ventricles to fill, and so forth.

The body of the book deals with what the authors term "encephalographic anatomy." Each portion of the gas-filled system is discussed in relation to the regional anatomy concerned. This approach to the study of the normal encephalogram is fundamental. The student of this field who attempts to build his experience from studying normal encephalograms alone will unnecessarily prolong the day of full comprehension of this field. The ideal approach to the mastery of this intricate field consists in the detailed study of "encephalographic anatomy" as presented in this monograph combined with a study of as many encephalograms as can be obtained.

HENDRIK J. SVIEN.

THE monograph, *The Abnormal Pneumoencephalogram*[1] by Leo M. Davidoff and Bernard S. Epstein, contains 506 pages with 695 illustrations on 289 figures. It is thus evident that the text is well illustrated, which is a prime requisite of a book dealing with such a subject. The contents of the book are divided into two parts, the first dealing with air studies in cases of brain tumor and the second part describing the pneumograms in cases of nontumorous lesions of the brain.

Part I is subdivided into two sections, the first dealing with neoplastic tumors and the second with nonneoplastic tumors. Inasmuch as the book was written not only for neurologists and neurosurgeons but for radiologists, psychiatrists, pediatricians, and internists as well, the subject matter includes a chapter on the fundamentals of neuropathology of brain tumors and brief descriptions of clinical syndromes and illustrative case histories.

The chapter breakdown in the section devoted to the pneumographic abnormalities associated with brain tumors is determined on a regional basis; that is, chapters are devoted to changes seen in tumors in the various regions such as the frontal, parietal, occipital, parasagittal, temporal and parasellar regions and so forth. Each chapter contains a review of pertinent literature and a statistical analysis of the authors' material regarding the type of tumor present, age range of patients, roentgenographic changes, and pneumographic changes. Although this statistical analysis makes for completeness of report, it is slow and somewhat difficult reading.

In the section devoted to nonneoplastic tumors, conditions such as subdural hematoma, brain abscess, aneurysm and hemorrhage, thrombosis, and so forth are discussed in relation to their pneumographic changes. A wide variety of conditions, including lesions of the septum pellucidum, agenesis of the corpus callosum, cerebral atrophy, platybasia and Arnold-Chiari malformation, tuberous sclerosis, and so forth, is considered in Part II dealing with nontumorous lesions of the brain.

Although in attaining proficiency in the interpretation of air studies, there is no substitute for critical study of a large number of pneumograms combined with a carefully correlated analysis of the surgical or postmortem findings, the student or practitioner will greatly aid and abet his efforts by reading this book carefully several times and then referring to the pertinent chapter in connection with each case he encounters.

HENDRIK J. SVIEN.

IN the extensive work, *The Kidney; Medical and Surgical Diseases*,[2] the author has covered the subject thoroughly. Although basically written from the viewpoint of structure, there has been a close correlation with the other preclinical sciences such as physiology and biochemistry. Further, he has clarified many debatable morphologic concepts and explained them in the light of clinical medicine. In other words, he has established a rebirth of the position of the pathologist in kidney lesions. Theories and unproved or untenable concepts are explained in the light of present day knowledge. No attempt is made to influence the reader by the mere expression of a belief in some unsubstantiated thought. This approach applies particularly in his treatment of the subjects of vascular disease and the relationship of the kidney to hypertension.

Dr. Allen has aimed at an integration of the study of the kidney as a unit. Following a brief introduction, he launches the context by giving the embryology, the anatomy, the normal and the abnormal physiology.

This is followed by a concise chapter on uremia which is a most thorough presentation and which this reviewer reread several times.

The malformations of the kidney, the renal pelvis, and the renal vessels are adequately presented. Diseases of the glomeruli and of the tubules are treated as separate chapters. There are satisfactory classifications and the definitive words are chosen to convey their exact meanings. It may be wise to indicate now that this reviewer is particularly pleased with the manner in which the author has categorized the entities in all chapters. It makes for simplicity in cataloging the material in one's mind.

There is a brief section on interstitial nephritis, one on specific infections, one on renal pelvic calculi, and one on renal rickets. Two of the major sections are devoted to disease of vessels (mentioned previously) and tumors. The latter includes the pelvis and pararenal tumors. Finally there are several pages covering histologic artefacts.

[1] THE ABNORMAL PNEUMOENCEPHALOGRAM. By Leo M. Davidoff, M.D., and Bernard S. Epstein, M.D. Philadelphia: Lea & Febiger, 1950.

[2] THE KIDNEY; MEDICAL AND SURGICAL DISEASES. By Arthur C. Allen, M.D. New York: Grune & Stratton, 1951.

There is an impressive number of photomicrographs and photographs in black and white. An occasional clinical photograph is included and there are also illustrated tables and drawings. Many of the author's associates have contributed microscopic material and full credit is given to them. Most of the specimens must have been collected by Dr. Allen over a number of years.

Both new and old articles are included in the references at the end of each chapter. Further due credit, in the selection of these, is given to the various scientists who have devoted much time to the study of the kidney.

The text should be a must in reading for all physicians, the general practitioner or the specialist, the graduate or the undergraduate student, the clinician, and the laboratory worker. It may well become the standard reference in the field of diseases of the kidney during the coming span of years.

M. C. WHEELOCK.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

MICRO-ARTERIOGRAPHY AND OTHER RADIOLOGICAL TECHNIQUES EMPLOYED IN BIOLOGICAL RESEARCH. By Alfred E. Barclay, O.B.E., M.D., D.Sc.(Hon.) Oxon., D.Sc.(Hon.) Manchester, F.R.C.P., F.F.R., F.A.C.R. Springfield, Ill.: Charles C Thomas, 1951.

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